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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XIV. APRIL, 1934 No. 56

Original Papers

CEREBRAL STRUCTURE AND MENTAL FUNCTION AS ILLUSTRATED BY A STUDY OF FOUR DEFECTIVES' BRAINS

By

R. J. A. BERRY and R. M. NORMAN

Stoke Park Colony

As the brain is undoubtedly the physical instrument of those human reactions which collectively form one's 'mind,' it appears reasonable to assume that the more closely the physical instrument can be correlated with its 'psychological' manifestations the greater will be our appreciation of the diversified causes underlying mental disorders of all kinds, and the greater our chances of arresting or even 'curing' them.

As a result of intensified research, improved technique and more accurate instruments of precision, our knowledge of cortical construction and cytology has within recent years been considerably advanced. But the various studies which have so largely contributed to this more detailed acquaintance with the development and neuronic connections and disconnections of the human cerebral cortex have not, perhaps, always been sufficiently, and in some cases not at all, correlated with the variations of behaviour observed during life. The converse appears to be equally true. In the present instance we propose to attempt this correlation for the brains of four mentally defective patients.

The four brains are those of the following cases:

1. A female imbecile (probably an idiot), age 12·1 years, with a mental age below 3 years.
2. A female imbecile, age 12·3 years, with a Merrill-Palmer mental age of 6·1 years.
3. A male Mongolian imbecile, age 23·9 years.

4. A feebleminded female, age 20·6 years, with a Binet mental age of 8·6 years, Porteus 8 years, and a combined Binet-Porteus intelligence quotient of 51.

These four brains have been selected from many others now in our possession, because they cover a representative and diversified series of juvenile and adult defectives. Their macroscopic features have thus been checked from others in the series. But as regards their microscopic examination, it has to be remembered that the histological investigation of any brain is a long and tedious process which demands for its successful elucidation both technical skill and considerable knowledge of brain structure. We therefore consider ourselves fortunate in having had the facilities for examining so completely as many as four. For purposes of comparison, and as a check on our own observations, we have also made use of normal brains, and particularly of the accepted facts of anatomy and embryology and of von Economo's detailed examination of the cytoarchitectonics of normal brains.

Although it is sometimes asserted that these and similar examinations of the brains and associated behaviour of certified mental defectives can throw little or no light on problems of the normal mind and its aberrations, our own experiences do not support such contentions. The legally certified defective only differs in degree—often very minute degree—from those not so certified, and the problems of his neurological ' make-up ' underlie all those of mind and its disorders. We are, therefore, led to hope that investigations such as this may be found of real practical utility to both psychiatrist and psychologist.

IMPORTANCE OF EMBRYOLOGY IN MENTAL SCIENCE

In the past, anatomical science has dealt faithfully and minutely with the names and descriptions of the sulci and gyri of the neopallial cerebral cortices of both anthropoids and man, but for obvious reasons has but little concerned itself with the origin, growth and development of the underlying causes, namely, the cerebral neurones. It would thus serve no useful purpose to attempt here any elaborate anatomical description of the fissures, sulci and convolutions of these four defective brains. On the other hand, though not perhaps universally recognized, all serious students of mental science would agree that there is an important relationship between the embryological development of the human brain and the subsequent mental reactions and/or aberrations of the adult. As this prenatal development is partly concerned with the formation of the necessary neuroblasts and neurones, which in their turn are responsible for the convoluting and fissuring of the originally smooth or lissencephalic neopallial cerebral hemisphere, the complexity of this fissuring, since it increases the total surface area of the cortex present, is an index of the potentialities of mental development possessed by the individual.

In the earlier months of intrauterine life the grey matter of the cerebral

cortex is spread out in a relatively thin layer, in which an active histogenesis of spongioblastic (neuroglial) and neuroblastic (neuronic) tissue takes place. Beginning with the fourth month, the pallial wall thickens rapidly, but there is as yet no manifestation of cortical fissuring. But as the cortical cell elements, especially the neurones, now commence to increase in numbers much more rapidly than the smooth pallial wall can provide accommodation for, the latter becomes thrown into folds. These folds become the future convolutions of the brain, and the intervals between them the fissures and sulci. The whole of this process is completed before birth. Even the secondary and tertiary sulci peculiar to the human brain are developments of the last two months of intrauterine life. Embryologically considered, it would appear that the cerebral convolutions and sulci result from cortical cell growth, that mentality depends on such cell growth, and that the foundations of these are laid down before birth. From such a standpoint it appears to be certain that the causes of the primary or endogenous amentias are to be sought in changes well established before birth, and not afterwards.

THE PRE- AND POSTCENTRAL CORTICAL AREAS

The three radial sulci—central, precentral, and postcentral—and the two gyri which, developmentally, are responsible for their appearance—namely, the precentral and postcentral—play such an important part in the mentality of every individual that they may be first briefly examined.

The postcentral gyrus forms one of the three most important strongly receptive areas of the cortex, and is largely granulous in construction. The precentral gyrus, on the other hand, is agranulous in structure, ' motor ' in function, and corresponds roughly to the ventral grey column of a spinal cord segment, whereas the postcentral gyrus is analogous to the ' sensory ' dorsal grey column of a similar segment.

Until about the end of the sixth month of intrauterine life there is nothing to differentiate the above mentioned sulci and gyri from the rest of the smooth neopallial cerebral wall. But as cell growth in the gyri concerned commences about this period to undergo a rapid proliferation, the pallial wall is thrown into folds, between which is developed the central sulcus. The central sulcus (fig. 1, no. 1) is therefore an infolding of cortex between two areas of different structure and function, the precentral and postcentral gyri. The cortical areas in question form the sensorimotor part of Flechsig's ' primary sense zones,' which he has shown have almost completed their development shortly after birth. It is thus obvious that whether the neurones of the receptor postcentral and effector precentral gyri be examined from the standpoint of their cytons (von Economo), or the myelinization of their axons (Flechsig), their main development is completed before, or just after, birth. If their development is not so completed, the future mentality of the individual must be profoundly modified, because it is impossible to

conceive any deprivation of such an important somæsthetic area as the postcentral gyrus leading to any other condition than a manifestation, in greater or less degree, of ' idiocy from deprivation of the senses.'

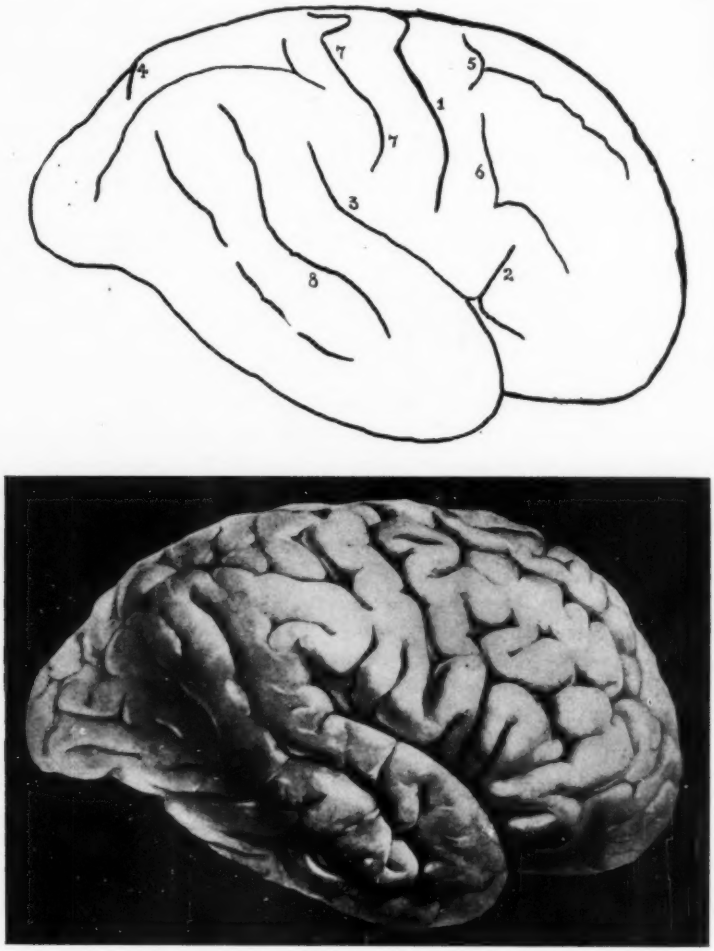
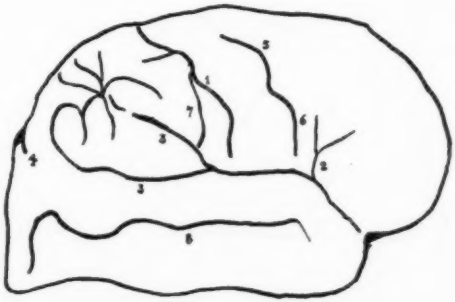


FIG. 1.—A Normal Brain.

Turning now to the four defectives' brains (figs. 2, 3, 4 and 5) with which this paper deals, it can be seen at once that they depart very considerably from those standards of normal development which are essential to the manifestation of a normal mentality (see fig. 1).

In fig. 2, the brain of a 12-year-old imbecile, the middle section of the postcentral sulcus (no. 7) cuts into the central sulcus (no. 1) and the vitally important receptive postcentral gyrus is so reduced as to be almost invisible on the surface.

In fig. 3 the inferior postcentral sulcus does not appear to have been developed at all, as the posterior limb of the fissure of Sylvius (no. 3) lies next to the central sulcus (no. 1)—an altogether abnormal arrangement.



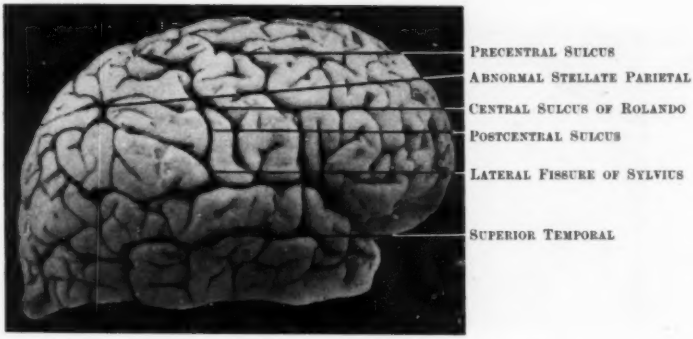


Fig. 2.—Imbecile No. 1.

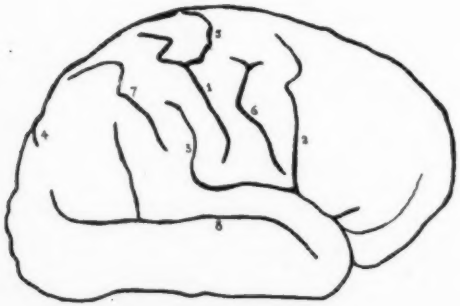
In fig. 4 the defect appears to have chiefly affected the upper part of the postcentral gyrus and the adjacent parietal lobe, which are both grossly lacking in anything approaching a normal development. The brain of this Mongolian imbecile displays the same general developmental errors as the other brains which belong to patients of no special clinical type.

In fig. 5, which is that of a higher mental grade than the others—namely, a feebleminded woman—the two areas in question, the receptor postcentral and effector precentral gyri, approximate more nearly to normality than do the others. As will be seen later, the chief developmental defects in this brain

occur in other regions, particularly the frontal operculum and the important parietal association area.

THE EFFECTOR PRECENTRAL GYRUS

The brains and their photographs show the same general gross departures from normality in the effector precentral gyrus and its limiting sulcus, the



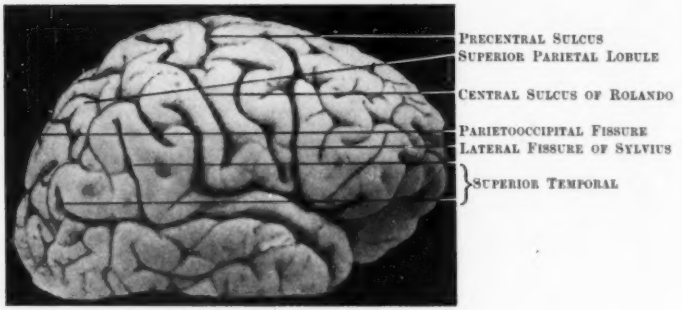


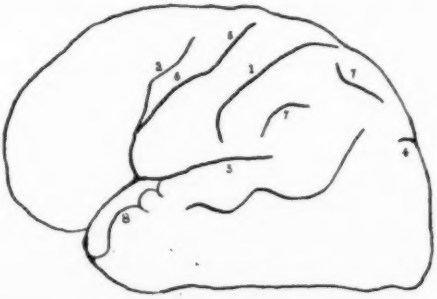
Fig. 3.—Imbecile No. 2.

precentral. In the adult Mongolian imbecile (fig. 4), for example, the inferior precentral sulcus (no. 6) appears not to have been differentiated from the Sylvian fissure (no. 2). In the second of the two imbeciles (fig. 3) the effector precentral gyrus is divided into two parts by a precentral sulcus (no. 5) which passes horizontally across it to cut into the central sulcus (no. 1)—an altogether abnormal arrangement.

In the feebleminded woman (fig. 5) there is a similar, but less complete, subdivision of the precentral gyrus into upper and lower parts, with an altogether abnormal inferior precentral sulcus of Y-shaped form (no. 6).

THE OPERCULAR CORTICAL AREAS

The cortical regions which, in the vicinity of the adult fissure of Sylvius, collectively form the anatomical opercula necessarily affect, by their varying development, the numbers of cortical neurones concerned in the future mentality of the individual. As the principal changes here concerned occur before birth, and not afterwards, they again confirm the view that endogenous



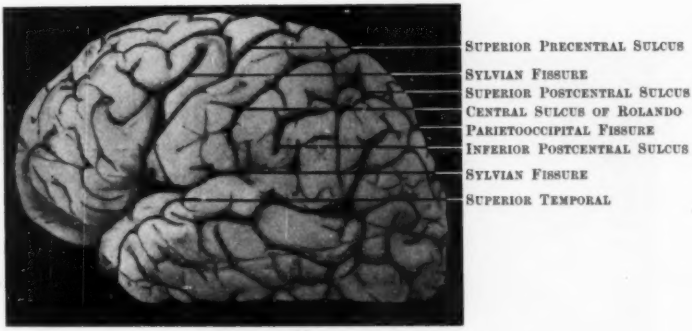


Fig. 4.—Mongolian Imbecile.

amentia is due to embryological errors of development, which are reflected in an imperfect fissuring of the cortex.

The development of the Sylvian fissure commences at the third month of intrauterine life, and is finally completed shortly after birth. Its formation is due to the fact that the neopallial wall in the region of the striate body does not enlarge as rapidly as the parts adjacent to it, thus forming a depression in the cortex known as the Sylvian fossa. As the neighbouring temporal, frontoparietal, and orbital portions become thicker, consequent on the increments in the numbers of their contained neurones, they form inrolling lips or walls, the socalled opercula, which finally cover in the retarded portion of insula. The lines along which the lips meet constitute the fissure of

Sylvius. The temporal and frontoparietal opercula are formed first, and the frontal and orbital later.

Variations in the form and arrangement of the two anterior limbs of the Sylvian fissure have long been known to, and described by, anatomists, but again such descriptions largely preceded more modern knowledge of the dependence of these variations upon the development and numbers of the underlying cortical neurones. The study of cortical cytology within recent years has, however, demonstrated that variations in the degree of develop-

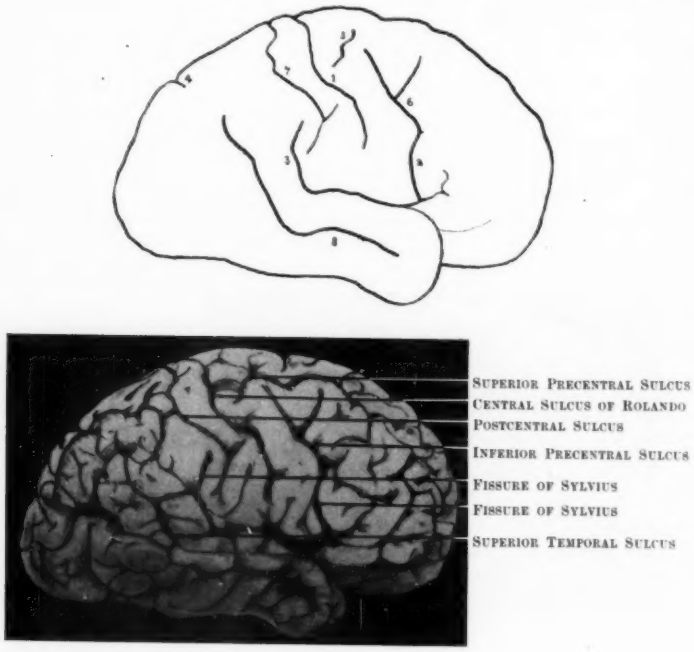


Fig. 5.—Feebleminded.

ment of the frontal operculum determine the shape of the two anterior limbs of the Sylvian fissure between which it lies. When well developed, this operculum separates them from each other so that they assume a U shape; when the operculum is less well developed the fissures assume a V shape; or if the operculum be so poorly developed as not to reach the Sylvian point, then the main limb of the fissure assumes a Y shape. In some forms of still greater arrest of development the anterior portion of the fissure of Sylvius is defective and the insula remains partly exposed. These macroscopic appearances of an undeveloped fissure of Sylvius and the dependent opercula mean the loss, in that brain, of many hundreds of thousands, and more probably

millions, of neurones with a consequent serious interference with the future mentality of the individual.

In the imbecile's brain which forms fig. 2, the frontal operculum is almost completely absent, and there is a small rudimentary Y-shaped anterior limb of the Sylvian fissure (no. 2).

In the imbecile's brain in fig. 3, the whole of the Sylvian fissure (nos. 2 and 3) is so abnormal as to make recognition of the anatomical parts a matter of speculation only. The frontal operculum lies within a U-shaped Sylvian fissure, but one of these limbs appears so unduly prolonged as to bisect the frontal lobe, while the frontoparietal operculum is both small and insufficient.

In the Mongolian imbecile (fig. 4), the insula is exposed and the frontal and frontoparietal opercula are almost nonexistent.

In the feebleminded woman (fig. 5), the frontoparietal operculum is distinctly better developed, but the frontal operculum, which is an offshoot of, in this case, the microgyric frontal association area, is quite abnormal. The anterior limb of the Sylvian fissure (no. 2) also assumes a very odd long-stemmed Y-shaped form, consequent apparently on its continuity with the inferior precentral sulcus (no. 6).

It is frankly impossible to study the opercular development of these defectives' brains without realizing that they connote a serious prenatal shortage of cortical neurones, and that such neuronic shortage must have exercised a profound influence in reducing the subsequent mentality to that of the legally recognized mental defective. Nor are these four brains singular in these respects, for the whole of a series of more than 30 defectives' brains now in our possession confirms the deductions herein made.

THE PARIETAL ASSOCIATION AREA

The important area—largely an associational one—known anatomically as the parietal lobe, reveals, in these brains, the same tendency to gross underdevelopment. The accompanying illustrations do not, on account of the convexity of surface concerned, reveal these macroscopic defects quite as clearly as do those already described, but the brains themselves leave no doubt as to the presence of the deficiencies now to be briefly mentioned.

In fig. 1, a normal brain, the usual appearances of the lateral surface of the parietal lobe are shown. The linear extent from the upper end of the central sulcus (no. 1) to the external limb of the parietooccipital fissure (no. 4) is of normal sufficiency, and indicates a parietal associational area adequate for a normal mentality. The other anatomical features, such as the subdivision into superior and inferior parietal lobules, and the further subdivision of the latter, by the upturned ends of the Sylvian (no. 3) and first (no. 8) and second temporal sulci, are seen to be normal and adequate for functional purposes.

But the four defective brains reveal a very different picture. The imbecile's brain in fig. 2 shows a parietal lobe which bears no resemblance to

any known normal arrangement. It consists only of a few convolutions arranged in a stellate form, from which it is difficult to understand how there can have been a normal mental functioning.

In fig. 3, another imbecile's brain, the convolutions in both the imperfectly developed superior and inferior parietal lobules are so primitive as to consist only of a few gyri so irregular as to make their naked-eye recognition a matter of speculation.

In the Mongolian brain represented in fig. 4, the superior parietal lobule is reduced to a thin bar of cortical tissue. The fissuring of the inferior parietal lobule is also irregular. The superior temporal sulcus (no. 8) is abnormal. Part of it appears to terminate in the anterior part of the Sylvian fissure (no. 3), so that it is doubtful if an angular gyrus is represented.

In fig. 5, that of a feebleminded adult woman, the inferior parietal lobule is relatively well developed, but the superior parietal lobule is little better than in the Mongol.

The importance of these obvious macroscopic manifestations of parietal underdevelopment consists in the fact that this area is largely associational in structure and function, and is set between the three important cortical primary receptive areas for general sensibility, vision, and hearing. The neuronic impulses set up by the stimuli acting upon these important combinations of ' senses ' are, or should be, synthetized, amongst other places, within the numerous associational neurones of the parietal lobe. Serious histogenetic errors within this area must, therefore, result in gross irregularities of the sulci and gyri concerned.

The foregoing description has applied only to three of the more important areas of the lateral surface of one cerebral hemisphere, but all show a similar tendency to prenatal lack of development, so serious as to be incompatible with a normal mentality.

OTHER EVIDENCES OF MACROSCOPIC CORTICAL MALDEVELOPMENT

Further evidence of this underdevelopment may be gauged from the following measurements :

	Frontooccipital length. Right cerebral hemisphere	Height	Upper border parietal lobe	Corpus callosum
	mm.	mm.	mm.	mm.
Imbecile	152	103	55	65
Imbecile	141	86	43	58
Mongolian	155	102	36	67
Feebleminded . . .	156	103	60	67
Normal (after Vierordt) .	167	106	65	81

The comparative figures for the corpus callosum alone furnish further evidence of the underdevelopment of these four defective brains, because the corpus callosum is a commissural bundle composed of axons having their cell bodies within the cortical grey matter of the two hemispheres and linking the latter together. If these cells are grossly reduced in numbers the corpus callosum should similarly be reduced in length and other dimensions.

CONCLUSIONS FROM THE MACROSCOPIC EVIDENCE

From this macroscopic examination it may be concluded that :

1. These brains are not, and never have been, sufficiently developed to give normal human reactions to normal human environments.

2. The errors responsible for this cerebral insufficiency occurred before birth, and, in these cases, were not due to disease.

MICROSCOPIC EXAMINATION

Sections were prepared and studied from eight representative areas, in all of which normal cortical structure is known, and a list of which is appended. In each of these eight areas an examination was made of the depth of the various layers and the number of cells per unit present in those layers. These measurements apply to the crowns of the gyri only. The left cerebral hemisphere was examined in Cases 1, 2 and 4 ; the right hemisphere in Case 3.

1. *Frontal Lobe.*
   (*a*) Precentral area (von Economo's area F.A.). Sections were taken from the upper part of the gyrus, near the border of the hemisphere.
   (*b*) Agranular frontal area (area F.B.). Sections were taken from the posterior end of the superior frontal gyrus.
   (*c*) Frontal association area (area F.D.). The anterior third of the superior frontal gyrus was examined.

2. *Parietal Lobe.*
   Superior parietal lobule (area P.E.).

3. *Temporal Lobe.*
   (*a*) The crown of the external surface of the superior temporal gyrus at its middle third (area T.A.).
   (*b*) Parasensory acoustic area (area T.B.). Sections were taken from Heschl's gyrus no. 1.

4. *Occipital Lobe.*
   (*a*) The visuopsychic area (area O.A.). Sections were taken from the convexity of the hemisphere at the anterior and upper part of the lobe.
   (*b*) The striate or visuosensory area (area O.C.).

THE RESULTS OBTAINED FROM LAYER MEASUREMENT AND CELL COUNTS

The depths of the cortical layers at the crowns of the gyri selected are shown in Tables A, and the number of cells per Hammarberg unit in these layers in Tables B.

TABLES A *

THICKNESS OF CEREBRAL CORTEX IN MILLIMETRES

1. *Precentral Area (Area F.A.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·29	0·25	0·21	0·29	0·18
L III .	1·08	0·72	1·19	1·02	1·5
L V . .	0·68	0·97	0·51	0·81	0·8–0·9
L VI .	1·23	1·36	1·02	0·98	1·0–1·5
Total .	3·28	3·30	2·93	3·10	4·5

2. *Agranular Frontal Area (Area F.B.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·34	0·34	0·25	0·32	0·22
L III .	1·27	1·10	1·02	1·10	1·5 +
L V . .	0·51	0·55	0·53	0·59	0·50
L VI .	0·85	1·40	1·49	1·27	1·25–1·50
Total .	2·97	3·39	3·29	3·28	4·0

* In these tables the normal figures are from von Economo and Koskinas' *Die Cytoarchitektonik der Hirnrinde des erwachsenen Menschen*, 1925, and the smaller English edition *The Cytoarchitectonics of the Human Cerebral Cortex*, 1929.

3. *Frontal Association Area (Area F.D.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·27	0·29	0·24	0·24	0·20
L II .	0·21	0·13	0·15	0·17	0·18
L III .	0·74	0·68	0·76	0·84	0·80
L IV .	0·21	0·17	0·21	0·19	0·21
L V . .	0·48	0·51	0·46	0·43	0·45
L VI .	0·93	1·19	0·98	1·01	0·90
Total .	2·84	2·97	2·80	2·88	2·8

4. *Superior Parietal Lobule (Area P.E.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·27	0·25	0·25	0·21	0·18
L II .	0·17	0·13	0·17	0·19	0·20
L III .	0·93	0·93	1·06	0·77	0·70
L IV .	0·27	0·25	0·21	0·24	0·40
L V . .	0·59	0·38	0·34	0·48	0·40
L VI .	0·98	0·81	1·19	0·72	0·90
Total .	3·22	2·75	3·22	2·61	2·6–3·0

5. *Superior Temporal Gyrus (Area T.A.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·27	0·25	0·29	0·27	0·22
L II .	0·13	0·08	0·11	0·13	0·1
L III .	0·81	0·68	1·02	0·85	0·95
L IV .	0·19	0·17	0·25	0·21	0·18
L V . .	0·42	0·55	0·51	0·51	0·50
L VI .	0·93	1·02	1·06	0·93	0·90
Total .	2·75	2·75	3·24	2·90	3·0

6. *Parasensory Acoustic Area (Area T.B.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·27	0·25	0·29	0·25	0·23
L II .	0·17	0·13	0·08	0·11	0·20
L III .	0·76	0·72	0·98	0·76	0·90
L IV .	0·25	0·21	0·34	0·21	0·35
L V . .	0·51	0·42	0·47	0·42	0·40
L VI .	0·85	1·02	1·06	0·81	0·80
Total .	2·81	2·75	3·22	2·56	3·0

7. *Visuopsychic Area (Area O.A.)*

	Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .	0·21	0·26	0·22	0·19	0·18
L II .	0·17	0·15	0·25	0·13	0·23
L III .	0·51	0·72	0·76	0·58	0·60
L IV .	0·21	0·19	0·21	0·24	0·20
L V . .	0·42	0·29	0·30	0·38	0·35
L VI .	0·73	0·68	0·64	0·53	0·70
Total .	2·25	2·29	2·38	2·05	2·3–2·6

8. *Visuosensory Area (Area O.C.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L I . .		0·21	0·19	0·23	0·21	0·19
L II .		0·06	0·08	0·05	0·04	0·12
L III .		0·29	0·21	0·28	0·29	0·27
L IV	*a*	0·21	0·17	0·21	0·13	0·17
	b	0·29 – 0·67	0·29 – 0·68	0·25 – 0·69	0·21 – 0·61	0·28 – 0·80 +
	c	0·17	0·22	0·23	0·27	0·17
L V .		0·21	0·25	0·25	0·25	0·27
L VI	*a*	0·21	0·17	0·21	0·21	0·25
	b	0·38	0·29	0·38	0·42	0·20
Total .		2·03	1·87	2·09	2·03	2·0–2·5

TABLES B

Cell Counts per Hammarberg Unit

1. *Precentral Area (Area F.A.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L III	*a*	13	21	27	22	30
	b	16	17	19	15	29
	c	12	7	12	12	15 +
	iv	16	16		13	12–15
L V	.	13	16	18	14	16
L VI	*a*	15	18	24	19	25
	b	13	14	16	14	15

2. *Agranular Frontal Area (Area F.B.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L III	ii	21	39	56	53	65
	a	19	12	27	21	30
	b	10	11	15	14	25
	c	11	7	9	13	18
LV	.	13	21	18	15	25
L VI		19	22	24	23	24
		16	13	15	17	15

3. *Frontal Association Area (Area F.D.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L II		48	47	72	62	75
L III	*a*	12	16	21	21	32
	b	10	14	20	14	16
L IV		47	44	58	59	85
L V	*a*	25	25	25	26	30
	b	11	13		14	15
L VI		27	24	27	27	30–35
		16	12	15	14	15–17

4. *Superior Parietal Lobule (Area P.E.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L II		83	64	67	76	110
L III	*a*	21	30	22	27	50
	b	15	19	16	16	25
	c	15	18	14	19	25
L IV	*a*	41	65	56	54	65
	b	50	71	52	56	80
L V	*a*	18	20	22	24	35
	b	14	18	16	16	17
L VI	*a*	27	27	24	34	21
	b	14	18	13	15	10

5. *Superior Temporal Gyrus (Area T.A.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L II	.	75	60	61	70	85
L III	.	18	15	14	17	40
L IV	.	66	50	39	61	100
L V	.	32	26	18	22	40
L VI	*a*	27	27	21	26	30
	b	18	13	14	17	15

6. *Parasensory Acoustic Area (Area T.B.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L II	.	73	73	70	94	80
L III	*a*	24	31	21	36	44
	b	16	18	17	32	30
	c	19	19	17	29	30
L IV	.	60	67	57	115	125
L V	.	28	38	33	38	50
L VI	*a*	30	34	29	42	40
	b	20	19	17	19	20

7. *Visuopsychic Area (Area O.A.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L II	.	74	70	74	80	80
L III	*a*	39	27	30	44	60
	b	26	15	24	27	45
L IV		126	74	86	88	150
L V	.	35	35	38	47	40
L VI	*a*	36	33	29	30	55
	b	18	13	11	16	12

8. *Visuosensory Area (Area O.C.)*

		Imbecile I	Imbecile II	Mongol	Feeble-minded	Normal
L III	.	68	62	62	66	100
L IV	*a*	99	106	85	90	150
	b	55	57	54	58	60
	c	158	136	130	159	220
L V	.	45	46	42	46	45
L VI	*a*	103	78	69	85	140
	b	22	24	16	20	20

From these tables it is evident that, in the majority of the areas examined, the total depth of cortex was not significantly diminished in these aments. Furthermore, the depth of the individual layers was, for the most part, equal to von Economo's normal figure, and in some instances was slightly in excess of it. Notable exceptions to the above generalization were, however, found in the agranular frontal cortex, especially in the supragranular layer of

IMBECILE 2. FEEBLEMINDED.

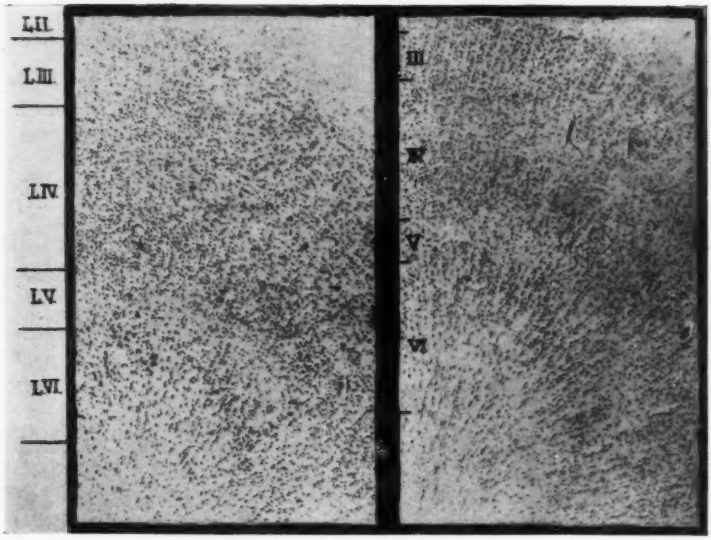


FIG. 6.—O.C. Visuosensory.

IMBECILE 1. MONGOLIAN. FEEBLEMINDED.

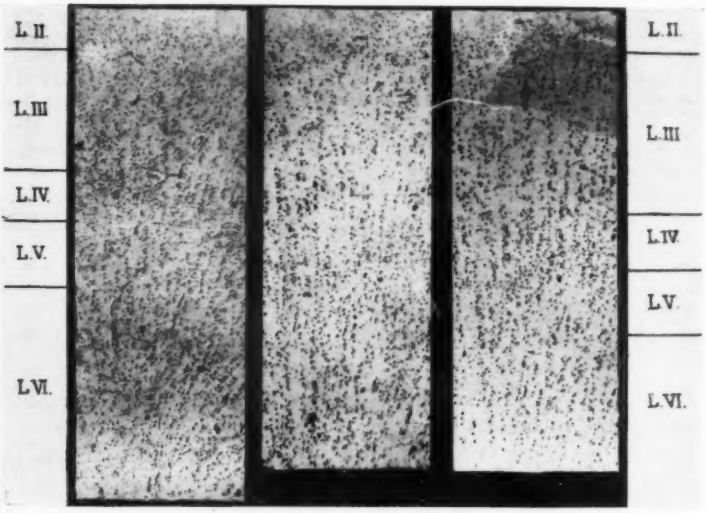


FIG. 7.—O.A. Visuopsychic.

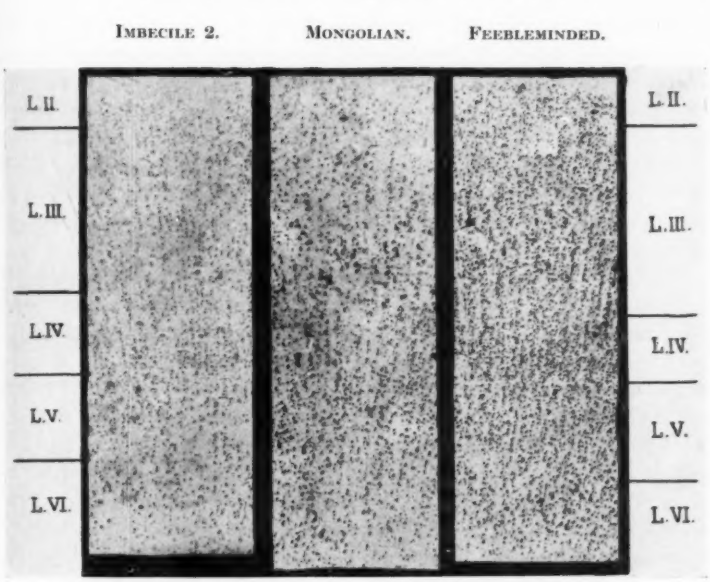


FIG. 8.—T.B. Parasensory Acoustic.

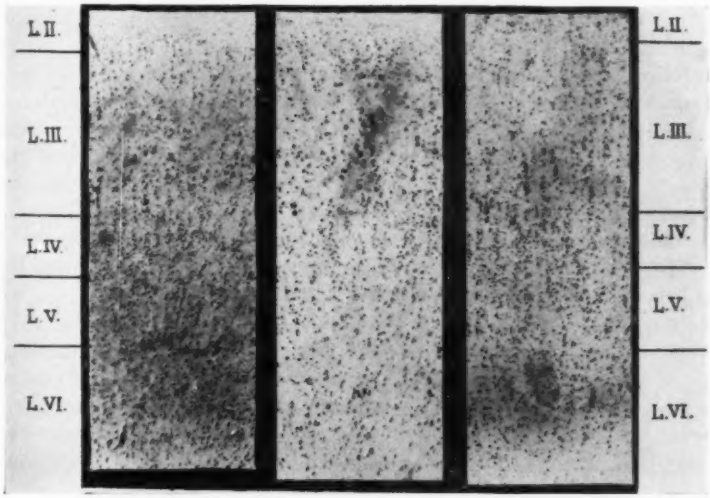


FIG. 9.—T.A. Superior Temporal.

pyramidal cells, L III (Tables A1, A2); in the granular layer, L IV, of the superior parietal lobule (Table A4); in the supragranular layer of the parasensory acoustic area (Table A6); and in the granular layer (L IV) of the visuosensory area (Table A8). The depth of the plexiform layer (L I) was almost invariably greater in these ament brains than in the normal, but as Campbell has pointed out, the variations in depth which this layer exhibits ' have to be viewed with caution, for not only do these alter in different parts of one gyrus, but measurements are falsified by the least obliquity of the section.'

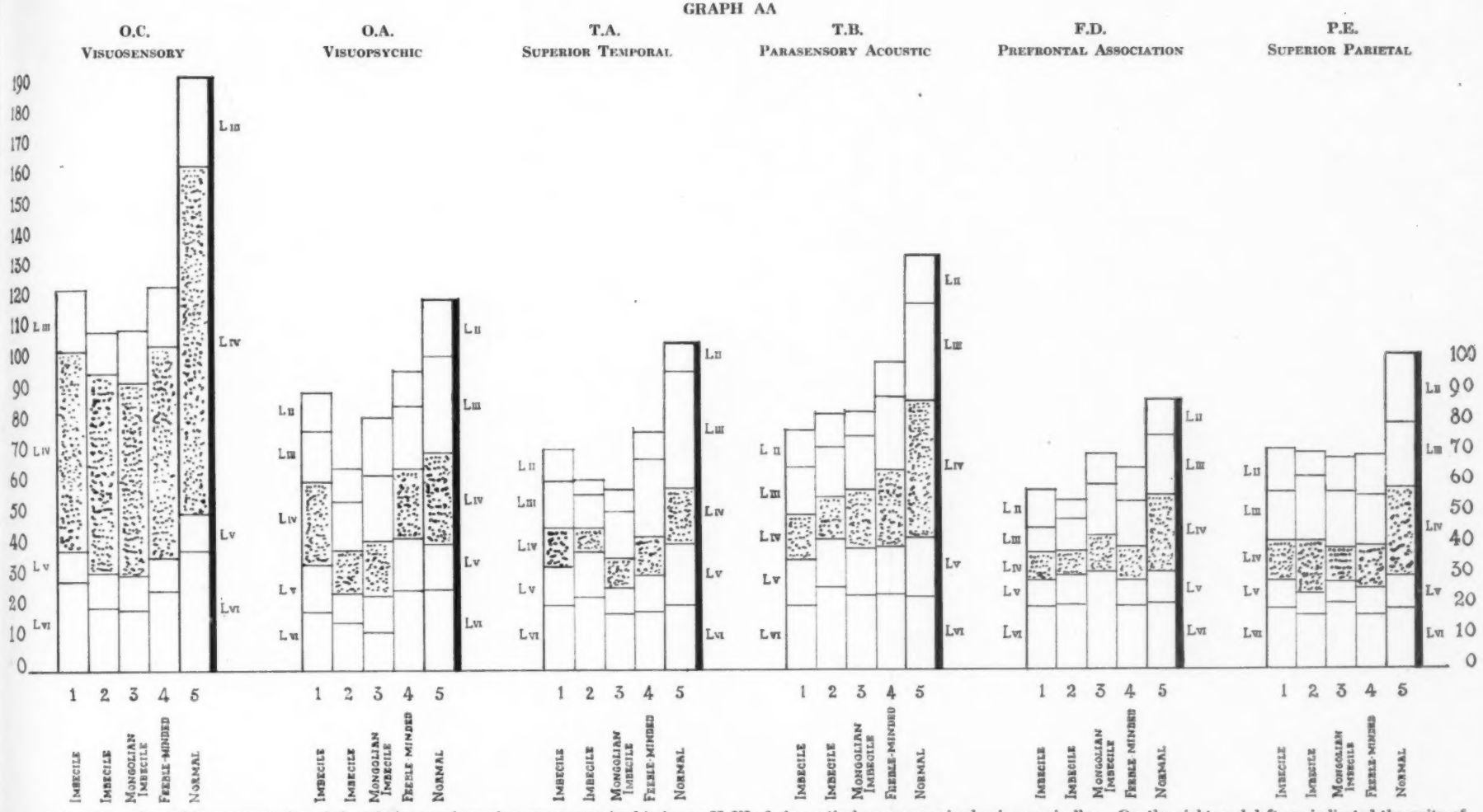
On the other hand, when the results of the cell counts (Tables B) are considered, it is clear that an entirely different state of affairs is present. In almost every instance the cell counts of the supragranular and granular elements were much below the normal. Lest it may be thought that some technical difference is responsible for this discrepancy, it must also be noticed that the cell counts of the infragranular cortex and especially of its deepest layer (L VI) approximate closely to the normal figures.

It is clear, therefore, that in order to get some idea of the neuronic efficiency of the part of the cortex examined by these methods, it is necessary to take both these factors of depth and number of cells into account. A measure of this total neuronic efficiency (so far as quantity and not quality of neurones is concerned) may be obtained by multiplying the depths of the cortical layers by the number of cells per unit these layers contain. The results of this procedure are set out in the three graphs.

In Graph BB is shown the relationship existing between the effector units of the supragranular cortex (pyramidal cells) and those of the infragranular cortex (fusiform and polymorphous cells). Graph AA shows the proportions in which all the cell layers are represented. In each case comparison with von Economo's normal is also shown. It is evident that the departure from normality is striking, and that the inferiority of these ament brains lies unmistakably in the supragranular and granular cellular development. The neuronic efficiency of the feebleminded patient with a Binet-Terman mental age of 8·6 years is also much closer to the imbecile level of the other cases than to the average normal level, but shows an appreciably better development of the parasensory acoustic area.

There are factors other than mere numbers of cells which have to be taken into account when individual differences in mentality are being discussed. One is the quality of the nerve-cells. Individual differences between the four aments are not well illustrated by a purely quantitative method, and this is in part due to the fact that in estimating the number of cells per unit in these four brains no distinction was drawn between immature neurones and neurones of good size and shape. In the cortex of the feebleminded patient the nerve-cells are generally of a better size and shape than in the imbecile cortices. In the latter, especially in the supragranular layers, instead of properly shaped pyramids, rounded or ovoid cells with scanty

GRAPH AA

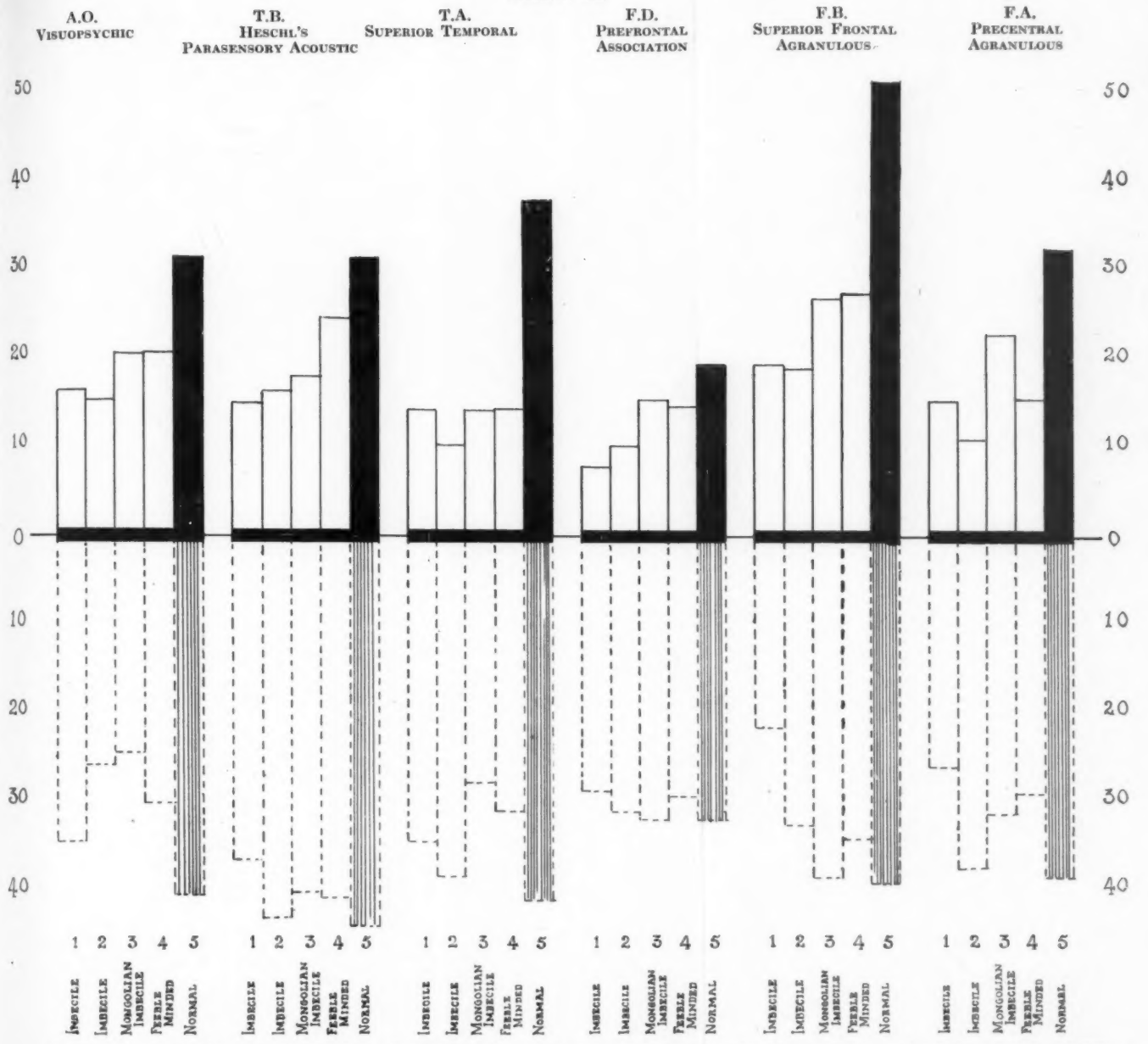


Graph AA.—A graphic representation of the relative numbers of neurones contained in layers II–VI of six cortical areas examined microscopically. On the right and left are indicated the units of measurement. These units have been obtained by multiplying the depth of each layer by the number of nerve-cells per Hammarberg unit it contains. In each case is shown the corresponding normal measurement as obtained by precisely the same method from von Economo's figures.

The granular cortex (L IV) of the several areas is dotted. The infragranular cortex (L V and VI) is represented below it, and the supragranular (L III, and II where present) above it.

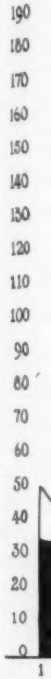
This qualitative method proves that, in every area, these four defectives are much inferior to normal in the development of the receptive granular, and the controlling supragranular, cortical layers, and also that the feebleminded patient is superior to the imbeciles in the visuopsychic, superior temporal, and parasensory acoustic areas.

GRAPH BB



Graph BB.—A graphic representation of the relative proportions of the cells of the supra- and infragranular layers (exclusive of L II and IV), as found microscopically in six cortical areas of the four defective brains. Units of measurement as in graph AA. The supragranular cortex is shown in the upper half of the graph above the zero line by vertical columns with continuous lines. The infragranular cortex is similarly shown below the zero line, but by columns with interrupted lines. For each area examined the standard of normality, as taken from von Economo's work, is shown on the right in solid black. The defective brains are displayed in the same order as in graph AA.

This graph illustrates the preponderance of the infragranular cortex over the supragranular, thus confirming other previous observations on the relative development of these layers. But it also brings out the further important fact that these defectives are lacking in an adequately developed supragranular cortical layer, to which is now attributed some of the higher functions of mind. In them, as the graph so clearly shows, the supragranular cortex is everywhere much below the normal standard in total numbers of pyramidal cells, and this is more so in some areas than others. From this microscopic evidence it is, therefore, clear that defectives suffer, not only from an underdeveloped supragranular cortex, but from an unequally developed one as well.


190
180
170
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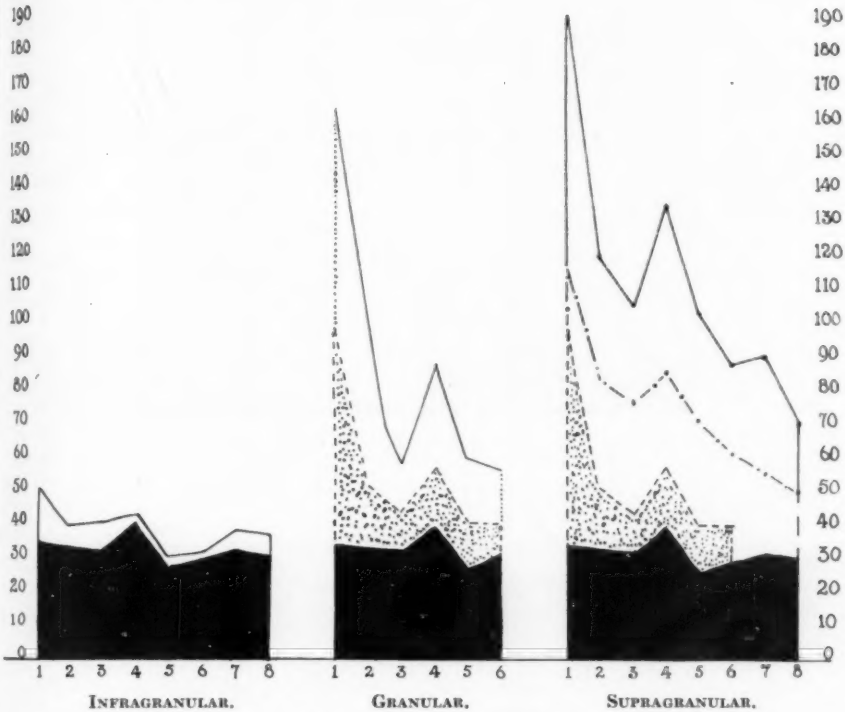
GRAPH CC



GRAPH CC.—In this graph the measurements of the four defectives' cortices have been averaged in each of the eight areas examined. The units of measurement are as in graph AA. These cortical areas are numbered 1 to 8 on the base line of the three figures, and are in the same order as mentioned in the text.

In the left-hand figure the infragranular layers of these defectives' cortices are shown in black. The normal excess above this level is indicated by an unshaded band.

In the middle figure the granular cortex of the defectives (dotted) has been added to the infragranular cortex (black). The standard for normal cortex, at this level, is shown above as an uninterrupted line.

In the right-hand figure the addition of the supragranular layer is shown as an interrupted line for the defectives, while the normal cortical level forms the upper boundary of the figure.

This graph illustrates the fact that cortical growth in the mental defective lags behind the normal, especially in the development of granular and supragranular layers. Physiologically expressed, these structural facts mean that the defective suffers from an inferior receptivity of stimuli and an inadequate power of integration and inhibition.

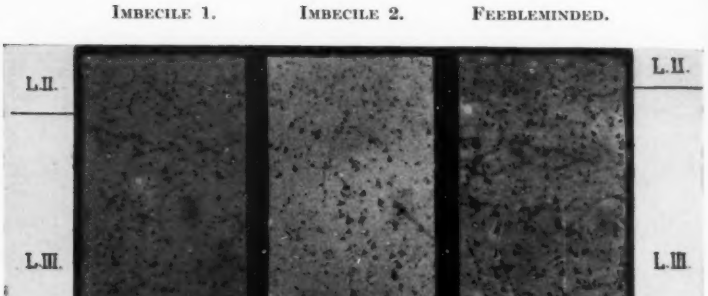


FIG. 10.—F.D. Frontal Association.

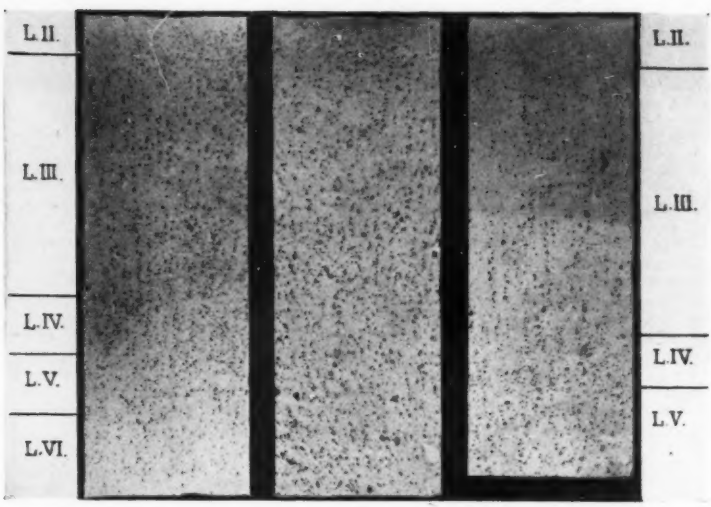


FIG. 11.—P.E. Superior Parietal.

cytoplasm and large pale nuclei are of frequent occurrence (see fig. 13b). A further peculiarity of such cells was their affinity for silver salts (see fig. 13c). These abnormalities in morphology and staining reactions are characteristic of the neuroblast, and it would thus appear probable that these illformed pyramidal cells are immature neurones.

Another factor to be considered when cortical structure is to be correlated with psychic activity is the general arrangement of the neurones. The microphotographs show that, with the exception of the frontal association area (fig. 10), the general architectural pattern of the cortex of the feebleminded woman is superior to that of the other cases. In her cortex not only is the horizontal lamination more clearly defined, but the vertical arrangement of the cells is more regular, thus indicating a greater wealth of intercellular fibres. Evidence for the latter conclusion is given by sections prepared by Bolton's iron alum method, which show that the myelinated fibres in these ament cortices are very inferior in numbers to those in a normal brain stained by the same method. This also shows that the radial bundles in the feebleminded cortex are larger than those in the imbecile. The lamination of the lower grade cortices was indeed often so confused that careful search had to be made along the crown of the section to find a part of the layer in a sufficiently demarcated state to admit of accurate measurement. Since mental life is the expression of the complexity and orderly development of cortical neuronic arcs, this factor of neuronic arrangement is of great importance.

When these factors of total numbers, quality, and arrangement of nerve-cells are reviewed in conjunction with the preponderance of the infragranular cortex present in all these cases, it is clear that the departure of these aments from the normal standards of mentality is explicable in terms of cortical structure. Furthermore, the two years' advantage in mental age possessed by the feebleminded patient over the imbecile is reflected both in the generally better cellular and architectural quality of the cortical areas examined as also in the superior development of the auditory zones.

SOME OTHER CORTICAL ABNORMALITIES DISCOVERED DURING THE COURSE OF THE INVESTIGATION

Amongst these were the following :

Abnormalities in the Frontal Association Areas.—A feature of these four brains is the presence of very poorly developed frontal association areas. The poverty of the two granular layers (L II and L IV) gives the sections the appearance of an intermediate (F.C.) type of cortex—namely, of that found in normal brains as a narrow strip lying between the agranular precentral areas and the well developed sesquilaminated frontal association area (F.D.).

In the frontal agranular areas (F.A. and F.B.) these four brains all show a considerable diminution in depth of, and number of cells in, the pyramidal cell layer III. In the Mongolian imbecile defective ' pyramidization ' (von

IMBECILE 2. MONGOLIAN. FEEBLEMINDED.

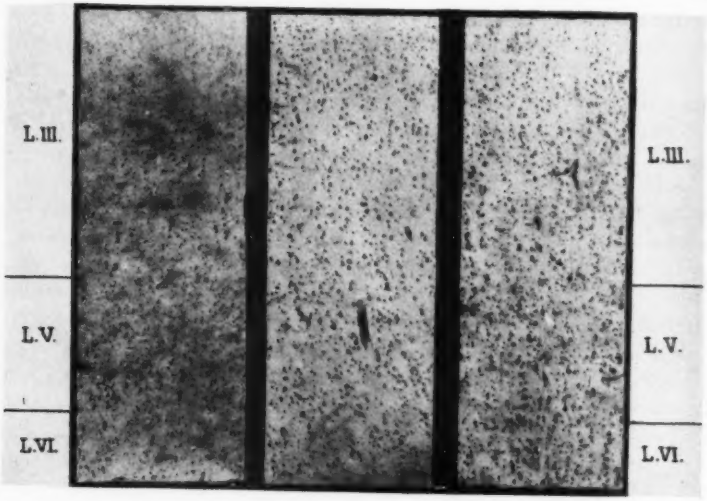
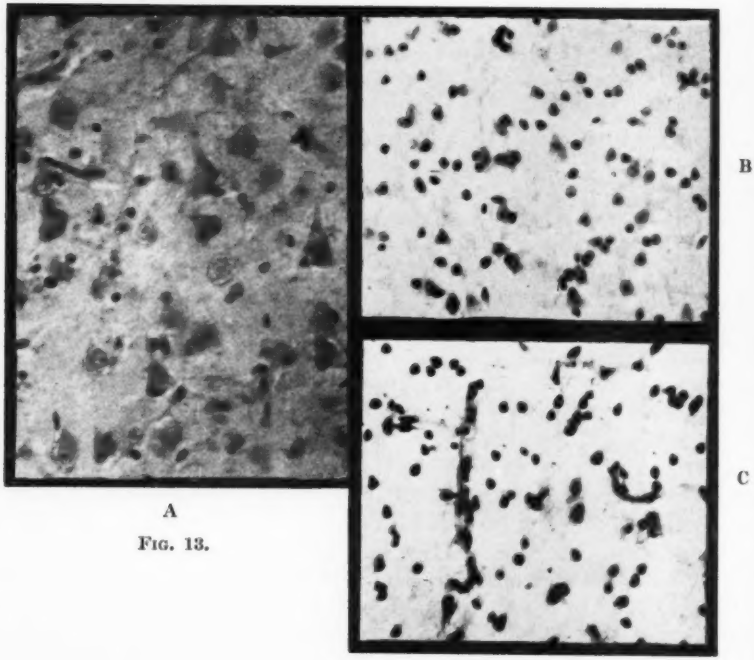


FIG. 12.—F.B. Frontal Agranular.

MONGOLIAN. IMBECILE 1.



A

FIG. 13.

IMBECILE 2. MONGOLIAN. FEEBLEMINDED.

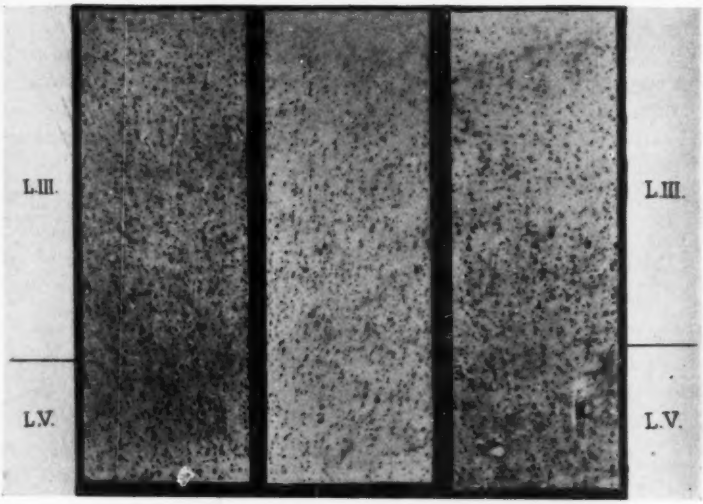


FIG. 14.—F.A. Rolandic Agranular Motor Area.

MONGOLIAN.

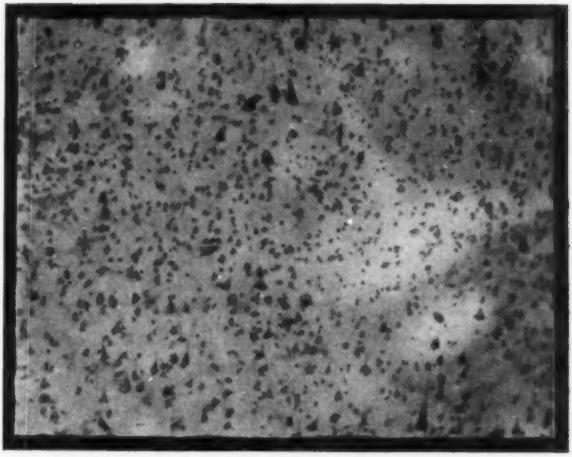


FIG. 15.—F.A. Defective Pyramidization.

Economo) of the cells in the lower part of layer III has occurred. That is to say, instead of a normal layer III (iv) consisting of medium pyramidal cells 20/10μ in size, a diffuse layer of granular and small pyramidal cells is present (see fig. 15). The Betz cells of the Rolandic motor area are smaller and not, as they should be in normal circumstances, an obvious feature of the sections. In our second imbecile the largest Betz cell seen in one section measured only 68 $\times$ 39μ, and in the feebleminded woman only 40 $\times$ 20μ.

In the Mongol (see fig. 13A) the frontal association area is also the seat of

FEEBLEMINDED.

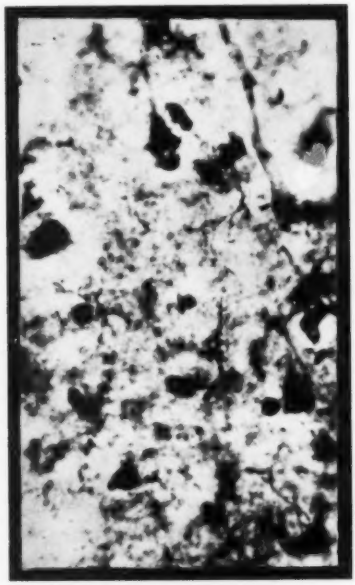


FIG. 16.—Microglia.

early degenerative changes, thus suggesting that these poorly developed pyramidal cells exhibited a diminished durability in the presence of the agonal stresses of toxæmia and raised temperature. This association of an undeveloped frontal lobe with an impaired mentality is also corroborated by Bolton, who, in his *Henderson Trust Lectures* (1933), says that the frontal lobe is ' the portion whose evolution renders possible the actual activity of the human mind . . .' and ' constitutes in fact the essential factor in vital activity.'

Abnormalities in the Parietal Association Areas.—In sections taken from the posterior end of the superior parietal lobule (area P.E.) the second imbecile shows an additional topographical abnormality. This part of the cortex had

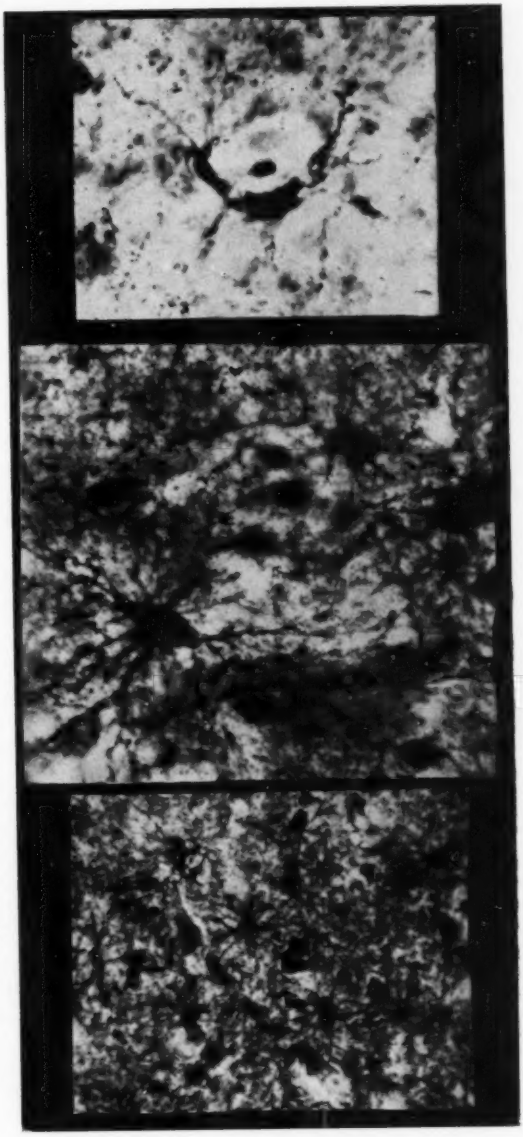


FIG. 17.—Neuroglia.

the easily recognizable features of that area of the superior parietal lobule which normally should lie immediately posterior to the somæsthetic sensory area (P.B.). These features consist essentially in the presence of giant cells in L III. But in this imbecile's brain sections taken from the anterior part of the parietal lobule show the ordinary superior parietal formation with no

FEEBLEMINDED.

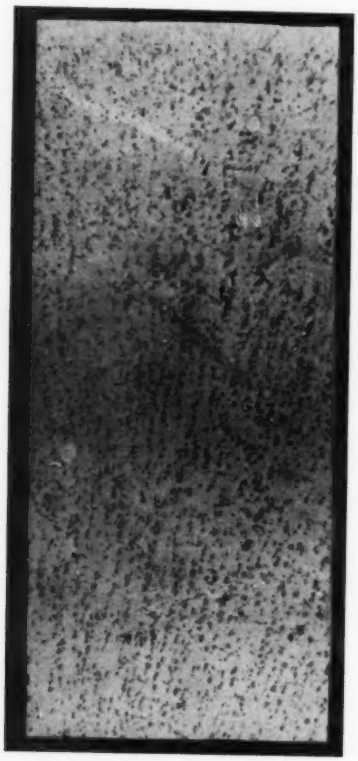


FIG. 18.—Inferior Parietal.*

giant cells in L III. This accidental finding suggests that a more elaborate mapping of the cortical areas in mental defectives would bring to light many minor aberrations of localization.

Abnormalities in the Temporal Acoustic Areas.—Another indication supporting the above suggestion that the abnormalities in the positions of

* This area, taken from the angular gyrus, was one of the most normal found. The depth and cell counts were:—L I 0·26; L II 0·26 (76); L III 0·82 (29); L IV 0·22 (102); L V 0·53 (20); L VI 0·82 (15). Normal: L I 0·22; L II 0.20 (95); L III 0·80 (25); L IV 0·35 (100+); L V 0·55 (18); L VI 1·2. (15).

recognized cortical fields may be of frequent occurrence in mental defectives is furnished by the second imbecile. In this case, four different blocks of cortex were removed from the gyri of Heschl of the left side in an attempt to locate the auditosensory area. Microscopic examination, however, revealed only cortical areas of parasensory type (T.B. or T.D.), thus showing that the auditosensory area had either developed in an abnormal situation or must have been very seriously reduced in extent.

Abnormalities in the Occipital Visual Areas.—In the same imbecile (case 2) the calcarine fissure was abnormally short. As measured from its junction with the parietooccipital fissure, it was only 0·5 cm. in length. It then divided into two terminal branches, the longer of which continued the direction of the parent stem towards the occipital pole for a distance of 0·9 cm., the shorter proceeding upwards for a distance of 0·3 cm. Thus the whole length of the fissure did not reach halfway along the medial surface of the occipital lobe, the total length of the latter being 3·2 cm. This anatomically abnormal short calcarine fissure is suggestive, of course, of a consequent diminution in the numbers of receptive granular visuosensory neurones. Microscopic examination of the area in question revealed no compensatory excess in either numbers of neurones or depth of cortex. It was, in fact, one of the worst of the series in both respects.

The poverty of a primary receptive sense zone should be reflected in the secondary, for development of the latter depends on correct stimulation by the former. It is to be expected that in this imbecile the visuopsychic area should reveal microscopic evidence of this lack of stimulation, and indeed it did so and was easily the most poorly developed of any of the four cases. Thus in the visuopsychic area of this imbecile L IV was wavy in appearance and varied considerably in total depth. Even the infragranular cortex shared in the general agenesia. L V was extremely poorly defined and L VI at the crown of the section showed many stellate instead of the fusiform cells which should be found in this situation.

The Agenesia of the Granular Layers.—Since these four brains all exhibit a measurable reduction in numbers of granular (receptive) neurones (L II and L IV) and do so in all parts of the cortex where these layers are normally present, there is every reason for supposing that this factor must, in its turn, have been responsible in part for the general cortical agenesia of pyramidal and other nongranular cells found in these brains. Ariëns Kappers and other authors hold that the Golgi Type II neurones of the cortical granular layer are strongly ' receptive and correlative ' in function, and that they transmit, sooner or later, the nervous impulses which they modify to the effector elements. It thus follows that if the effector cells receive only an impaired stream of impulses, their development will tend to remain at a low level. Thus the lack of numbers of these effector cells may be attributed to a primary agenesia, and their poor quality to lack of stimulation. Clinical examination showed that these patients were not defectives from ' deprivation of the

senses ' in the ordinary sense of the term, but were so because a maldeveloped cerebral cortex is incapable of ' using ' the stimuli faithfully transmitted to it from the environment.

CONCLUSIONS

From the microscopic examination described in the text, it may be concluded that :

1. There is a significant reduction in the numbers of supragranular and granular neurones, though the total depth of cortex at the crowns of the gyri is not appreciably diminished except in the agranular frontal cortex.

2. These cortices are of inferior neuronic construction. The brain cells are irregularly arranged, tend to be small in size and are often of abnormal shape. Further, topographical variations may be present in low grade cortices, as has been noted in at least one of these imbeciles.

3. The cortex of the feebleminded case mainly differs from the imbecile in the more normal arrangement and shape of its cells. It is thus not improbable that the difference in cortical structure between high and low grade aments is one of degree.

SOME CLINICAL AND GENERAL CONSIDERATIONS

Both macroscopically and microscopically the relationship of these brains to the relative mental states of the patients is so obvious as to require no further demonstration. Their undeveloped condition sufficiently accounts, without any superadded psychological or other theoretical explanation, for the admitted legal mental deficiency during life. The imbeciles, for example, could not write, read, count, or attend to their own requirements, notwithstanding that much time had been devoted to their socalled ' educational ' and manual training.

As the cerebral irregularities displayed by these cases are prenatal in origin, and incapable of structural alteration by postnatal treatment, it would appear to be futile to expect ' cures,' for no form of educational, psychological, or occupational therapy can supply brain cells where nature has denied them. More stable habits can, of course, be established partly as the result of a natural chronological growth and partly by persistent and constant training, which in many of these aments—including some of the lower grade feebleminded defectives—is of little more value than a filling in of time between eating and sleeping.

The discovery in many defectives of a clinical syndrome, or of some condition simulating one, is sometimes assumed to be evidence warranting a diagnosis of secondary or exogenous amentia. Organic nervous disease can be, and often is, engrafted on to a preexisting endogenous amentia, and the latter condition naturally tends to increase the incidence of nervous degenerations amongst defectives, in whom they are, indeed, very common. Thus the first imbecile of the present series displayed during life the clinical

picture of extrapyramidal paralysis of Parkinsonian type, and a characteristic paucity of nerve-cells was discovered later in the substantia nigra. A moderate degree of gliosis and an increased number of bloodvessels indicated a chronic degenerative process. Yet the grossly abnormal fissuring of the brain and the microscopic deficiency of cortical neurones are those of the primary ament.

Any considerable reduction in the number of cortical neurones is attended by a corresponding reduction in the numbers of the medullated axons of the associational, commissural, and projection systems. The corticipetal (receptor) and corticifugal (effector) axons of the last named system pass between the cerebral cortex and practically all other parts of the nervous system. The diminished size of the corpus callosum, and more particularly the underdevelopment of the post- and precentral convolutions, as well as that of the Betz and other pyramidal cells, suggest the consequential fact that, in these cases, other parts of the central nervous system must have shared in the lack of development so clearly indicated by the cerebral cortex. Such underdeveloped nervous systems necessarily function so imperfectly as to give rise to aberrant neurological phenomena which, whilst occasionally simulating the syndromes of disease, for which they are readily mistakable, are in reality of developmental, not pathological, origin. In a previously normal individual precisely the same reduced level of neuronic efficiency may be reached as a result of genuine disease processes. Of the many lessons which the defective brain and nervous system of the ament have to teach, one is, apparently, that syndromes of neurological disorders closely resembling one another may, in reality, have two quite different origins, the one developmental, the other pathological—facts which have a possible bearing on both diagnosis and treatment.

That the ament, with his considerably reduced expectation of life and his consequent much earlier senility—that is, a physiological degeneration of nerve and other cells—frequently combines both causes within his own person, occasions no surprise. It is, indeed, not improbable that these facts also apply, in lesser degree, to patients other than defectives of inferior neuronic development who develop phenomena resembling neurological syndromes more usually caused by organic disease. If so, this would be in accordance with the clinical conception of abiotrophy applied to many of the familial degenerative syndromes of the central nervous system and defined by Karsner as follows: 'this socalled nervous abiotrophy seems to occur in cases in which, owing to hereditary or other inherent defect, certain localized regions are unusually susceptible to deterioration.'

All the cells of the human body—somatic, reproductive, and nervous—result from repeated subdivision of a fertilized ovum. In amentia there is a reduction of the numbers of nerve and, presumably, other cells. Primary mental deficiency is thus a genetic manifestation of the inability of a fertilized human ovum of usually impaired stock to undergo a correct cellular division

and multiplication. Hence the physical basis of so many mental and nervous disorders and the potent influence of heredity in their production and transmission.

APPENDIX

1. *Note on the Neuroglia and Microglia.*—The cortices of one of the two imbeciles, the Mongolian imbecile and the feebleminded woman, were examined by Cajal's gold sublimate method and Rydberg's silver-diamino-carbonate and combined gold-silver methods for the study of the neuroglial and microglial elements. The distribution of the protoplasmic astrocytes (see fig. 17c) suggests that the thickness of these cortices, which is surprisingly normal when compared with the neuronal poverty they display, is due to the ramifications of glial processes and to an increased number of the glial cells. These fill out the spaces between the scattered and numerically diminished neurones. The microglial cells were generally numerous, of the somewhat swollen (see fig. 17a) and occasionally present as long bipolar forms.

2. *Technique.—Fixation.*—Ten per cent. formol saline solution was injected into the cisterna magna and through the orbits into the interior of the skull immediately after death. After removal, the brain was suspended in the same fixative and on the third day small incisions were made to allow the fluid freer access to the ventricles.

Methods used in Measurements and Cell Counts.—The blocks removed from the cortical areas to be examined have to be cut at right angles to the surface of the brain and to the direction of the gyrus. Two factors tending to flatter the defective now become operative. The first is that the macroscopically better formed gyri are selected, since they are easier to cut; the second is that any obliquity of cutting in the process of removing the block of tissue will increase the depth of the layers to be measured.

Sections were cut on a freezing microtome at 25μ and stained by 1 per cent. toluidin blue. It was found best to fix each section to an albuminized slide owing to the tendency of the section to shrink in the xylol stage of the staining process. The depth of the cortical layers at the crown of the section was measured by means of an eyepiece micrometer ($\times$ 10) using a Leitz No. 3 objective. For the cell counts an Ehrlich eyepiece was used in conjunction with a No. 6 objective, the average of at least twenty contiguous fields being taken. This result was then converted into the Hammarberg unit—namely, the number of cells present in 0·1 cubic millimetres.

EXPLANATION OF THE ILLUSTRATIONS

FIG. 1.—Photograph of a normal brain for comparison with those of the four defectives.

FIGS. 2–5.—Photographs of the four defective brains described in the text.

FIGS. 6–12 and FIGS. 14 and 18.—Microphotographs of different cortical areas of the defective brains showing the cellular variations of structure. Toluidin blue. $\times$ 48.

FIG. 13.—(*a*) Cortical neurones of the frontal association area of the Mongol, showing early degenerative changes. Toluidin blue. $\times$ 360.

(*b*) Lamina II of the visuopsychic area of imbecile no. 1, showing numerous rounded immature neurones instead of fine pyramidal cells. Toluidin blue. $\times$ 360.

(*c*) As in (*b*), but stained by Rydberg's silver-diamino-carbonate method. $\times$ 360.

FIG. 15.—Section of the Rolandic motor area (normally agranulous), showing the presence in the Mongol of an abnormal granular layer. Toluidin blue. $\times$ 80.

FIG. 16.—Microglia in the Rolandic motor cortex of the feebleminded woman. Combined gold-silver method. $\times$ 360.

FIG. 17.—(*a*) As in fig. 16. $\times$ 800.

(*b*) Protoplasmic astrocytes in L III of the Rolandic motor cortex of the feebleminded woman. Cajal's gold sublimate method. $\times$ 800.

(*c*) Protoplasmic astrocytes in L III of the Rolandic motor cortex of the Mongol. $\times$ 360.

THE DEPTH AND RATE OF RESPIRATION IN NORMAL AND PSYCHOTIC SUBJECTS

By

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INTRODUCTION

In recent years an increased interest has been taken in the respiration of psychotic subjects. Many observations pointed in this direction as possibly a fruitful method of approach to the physical side of mental disease. Golla, Mann and Marsh [1] in 1928 found that the response of these patients to the inhalation of CO_2 is different from that of healthy subjects. That the well-known effect of CO_2 in temporarily abolishing catatonic stupor [2] is not due to mere fright, is borne out by recent work [3] by Paterson and Richter, who showed by exact measurements that the catatonia-like state of monkeys poisoned by bulbocapnine is increased by small doses of the drug and abolished by large doses.

The following report describes the result of an investigation of the depth and rate of respiration in schizophrenics and melancholics.

It had long been remarked clinically that the subjects of dementia præcox appear to breathe more shallowly and more rapidly than normal subjects. The exact measurement of the depth and form of respiration, however, has been beset by difficulties. Most observations recorded in the literature have been made by means of a stethograph, that is to say, a rubber tube of some kind fixed round the thorax or abdomen. As such an apparatus may direct the subject's attention to his breathing, the value of the results obtained may be vitiated. Moreover, such a method cannot record accurately the magnitude of each respiration, although observations of the magnitude of the respiratory movements in psychotics taken by this method have been published.[4,5] Another objection is that the stethograph is only measuring one part of the respiratory mechanism, and at any moment the respiration may change from thoracic to abdominal or vice versâ. In the course of one of my experiments in which records of the thorax and abdomen were being made simultaneously, the movements of the abdomen became smaller and those of the thorax greater without altering the amount of air respired, as measured simultaneously by the plethysmograph. The excursion recorded therefore in one case by this method cannot be compared quantitively with that recorded

in another, or even with the excursion in the same individual at different times.

TECHNIQUE

The apparatus described by Golla and Antonovitch [6] disposes of these difficulties. It consists essentially of a wooden box with a removable lid and front piece, both of which can be clamped firmly into position by means of a handscrew. The subject for experiment sits in the box and only his head projects through a hole in the roof. The box is air tight and the opening for the neck is occluded through the subject's wearing an inflatable rubber collar, to which an 'apron' is attached all round. The latter is clamped between two boards in the roof of the box. These collars are made of varying sizes. The air space in the box communicates with the recording plethysmograph by a wide tube.

The recorder itself is of the simple float pattern, rectangular, and having a capacity of about 4 litres. The float is balanced on knife edges at its posterior end, the edges resting in corresponding notches on the wall of the water bath.

With this apparatus I have recently taken over 300 records of patients and normal subjects at the Maudsley and Colney Hatch Hospitals with a view to studying the form, rhythm, reaction to stimuli, etc., of psychotics in comparison with normal subjects. In the present paper, I propose merely to record the results of observations on the depth and rate of respiration. The results of the other studies will form the subject of a further communication.

Several precautions were taken to keep the conditions uniform throughout the experiments. No external stimuli were allowed to disturb the patient. He merely sat quietly in the box, and was unable to see any of the recording apparatus. He was unaware that his respiration was being examined. His attention was therefore not directed towards his respiratory movements, a circumstance which would ipso facto have rendered the results less valuable. Factors which might interfere with the processes of metabolism were kept uniform. The subject sat still until he had become accustomed to his environment. If he was obviously alarmed, a fact which seldom occurred, the experiment was abandoned. No record was taken after the subject had been active physically, or soon after a meal. A minimum of clothing was employed in order to prevent undue warmth inside the box.

RESULTS

The accompanying table shows the results of these investigations. In this series 217 subjects were examined, including 44 normal men and 18 normal women. These were members of the staffs of the Maudsley and Colney Hatch Hospitals, and to them my thanks are due; 85 schizophrenic males and 36 schizophrenic females were examined. Most of these were

suffering from a considerable degree of dementia. By way of comparison, six males and 26 patients who were melancholic but not demented were examined.

TABLE I

Number	Class	Mean area of body surface in sq. metres	Mean vol. of tidal air in c.c.	Mean rate of respirations per min.	Mean vol. of respirations per min. in litres	Presumed size of dead space in c.c.	Mean effective ventilation per respiration in c.c.	Mean effective ventilation per min. in litres
	MALES							
44	Normals . .	1·815 ± ·012	572	16·77 ± ·329	9·59	180	392	6·57
85	Schizophrenics .	1·592 ± ·008	442	19·61 ± ·293	8·67	180	262	5·14
6	Melancholics .	1·630	623	18·67	11·65	180	443	7·84
	FEMALES							
18	Normals . .	1·578 ± ·016	429	18·61 ± ·494	7·98	137	292	5·43
36	Schizophrenics .	1·509 ± ·020	339	22·67 ± ·363	7·68	137	202	4·58
19	Melancholics .	1·517 ± ·020	402	19·55 ± ·685	7·86	137	265	5·18
8	Normals (dozing state) . .	1·578	491	13·50	6·62	137	354	4·79
8	Normals (sleeping state) . .	1·578	317	15·00	4·76	137	180	2·70

When one compares the amount of air respired as between two groups, the size of the subjects must be taken into consideration. One would expect a large individual to breathe more deeply than a small one. The factor used by physiologists to make possible a comparison of the oxygen consumption of two individuals of different size is the area of body surface, and this has been used in the present study. It was calculated from the height and weight of each patient by means of Boothby and Sandiford's nomograph.[7] The mean size of each group is shown in the first column in square metres of area of body surface. The second column shows the mean volume of the tidal air of each group measured in cubic centimetres. The third column shows the mean number of respirations per minute for each group. When this is multiplied by the figure giving the tidal air we obtain the volume of air breathed per minute, here given in litres in the fourth column.

Looking at the volume of air respired (tidal air) as recorded in the second column, one finds that the figure for normal males is a little higher than that usually given in text-books for subjects at rest. The usual figure is 500, but the present figure is 571. The reason for this is that the subject was sitting, not lying, and that the temperature inside the box was rather higher than normal.

In order to allow for the fact of difference in size, the mean size of each group, normals, schizophrenics and female melancholics, was calculated. The probable error and standard deviation were worked out together with the correlation coefficient of volume of respiration with area of body surface.

TABLE II

Area of Body Surface in Square Metres

	Mean	Standard deviation
MALES		
Normals . . .	1·815 ± 0·012	0·1138
Schizophrenics . .	1·592 ± 0·008	0·1153
FEMALES		
Normals . . .	1·578 ± 0·016	0·1021
Schizophrenics . .	1·509 ± 0·015	0·1372
Melancholics . . .	1·517 ± 0·020	0·1315

Comparison of Size of Normals and Schizophrenics.—It will be noticed in Table II that the mean area of body surface of male schizophrenics (1·592) is very much smaller than that of normals (1·815), the difference being significant. For females the schizophrenics have a mean area which is smaller than for normals to an extent which is just significant, the difference being three times the probable error (0·069 ± 0·02).

TABLE III

Volume of Tidal Air in Relation to Size of Subject

	Volume of tidal air in c.c.	Standard deviation	Correlation coefficient with area of body surface
MALES			
Normals . . .	572	143	$r = + 0·2216$
Schizophrenics . .	442	79	$r = + 0·1096$
FEMALES			
Normals . . .	429	74	(Too few cases)
Schizophrenics . .	339	52	$r = - 0·0419$
Melancholics . . .	402	57	(Too few cases)

For males, the schizophrenics have a much smaller mean volume of tidal air and also a variability which is much less than for normals. Part of the difference in the means results from their smaller size, but this can be taken into account since the correlation of volume of tidal air with area of body surface is known. The volume of tidal air which normal subjects with an area of body surface of 1·592 square metres would be expected to have is 510 c.c. Actually, the male schizophrenics had a mean volume of 442 c.c. Hence the difference between the actual and expected is 68 c.c. As its probable error is ± 15·6 c.c., the difference is more than four times its probable error after correction for difference in size, and is therefore significant.

For females there is no sensible correlation between volume of tidal air and area of body surface, and therefore there is no need to correct for the slight difference in mean area of body surface. The mean volumes may therefore be compared as they stand. For female schizophrenics the difference from normals is $90 \pm 13{\cdot}0$ c.c. Since the difference is about seven times its probable error, it is certainly significant.

Rate of Respiration.—Since there is no correlation between rate of respiration and area of body surface, there is no need to correct for difference in area of body surface. For males the rate for normals (see Table I) was $16{\cdot}77 \pm 0{\cdot}329$, and for schizophrenics $19{\cdot}61 \pm 0{\cdot}293$. The difference is $2{\cdot}83 \pm 0{\cdot}441$, which is six times its probable error, and is therefore certainly significant.

For females, the normals show a rate of $18{\cdot}61 \pm 0{\cdot}494$, while that of schizophrenics is $22{\cdot}67 \pm 0{\cdot}363$, a difference of $4{\cdot}06 \pm 0{\cdot}613$. Since the difference is over six times the probable error, it is again certainly significant.

From these results then it appears that schizophrenics breathe more shallowly and more rapidly than normals.

The question arises whether rapid shallow breathing is as effectual a method of ventilating the lungs as deep slow breathing. At first sight the fact that the total volume breathed per minute in each case is about the same might seem to show that each is equally effective, but certain considerations are against this view.

The degree to which the lungs are effectively ventilated depends on the amount of the socalled dead-space, or the space within the respiratory passages which prevents a certain proportion of the tidal air from reaching the alveoli. It is still a matter for controversy among physiologists as to how much the dead-space varies in size under different conditions of breathing. It is probable that it varies little, and if this is so, increased depth of respiration is much more important than increased rate, to bring about the effective ventilation of the lungs.

The following figures from Roaf's * *Physiology* show how, if the depth

Volume of respiration	Dead-space	Effective ventilation (obtained by subtracting the second from the first)
300 c.c.	172 c.c.	128 c.c.
400 c.c.	172 c.c.	228 c.c.
500 c.c.	172 c.c.	328 c.c.
700 c.c.	172 c.c.	528 c.c.
900 c.c.	172 c.c.	728 c.c.
1,200 c.c.	172 c.c.	1,028 c.c.
4 times		8 times

* A. E. Roaf, *Textbook of Physiology*, 1924, p. 237.

of respiration in one subject is four times that of another, the effective ventilation of the former is eight times, not only four times, that of the latter, given that the dead-space and rate of respiration are the same in both cases.

If, however, the rate of respiration is four times as fast in one subject as in another, where the depth is the same, then the effective ventilation is only four times as great.

Supposing, therefore, as is not unlikely, that the dead-space of the schizophrenic patients is of the same magnitude as that of normals, then the fact that they breathe more shallowly will not be compensated for by the greater rate of respiration.

The next three columns in the table represent an attempt to take into consideration the dead-space. If we do this, we shall obtain a more accurate idea of how much less effectively the lungs are ventilated in schizophrenics than in normals. The theoretical size of the dead-space in a normal subject, whose tidal air is, as in this group, 572 c.c., is 180 c.c., according to a well-known theoretical formula,* if there is an average percentage of O_2 and CO_2 in the alveolar air. If we suppose for the present purpose that the dead-space in the different classes of male subjects under investigation is about the same, then we shall get as figures for effective ventilation those shown in the table. In the group of normal males the mean tidal air is 572 c.c. If we subtract from that figure 180 c.c. of dead-space, we obtain the amount of air passing into the alveoli. If this is multiplied by the rate of respiration per minute, 16·77, we get 6·57 litres, or the effective ventilation per minute.

In the second line, the dead-space for schizophrenics is presumed to be the same as for normal males, namely, 180 c.c. If this is so, and it is probably about the same, then the effective ventilation, per respiration, is 262 c.c., and, per minute, 5·14 litres. This figure is only 78 per cent. of the normal figure, whereas if the dead-space were neglected the respiration would be 90 per cent. of the normal.

Similarly for females, we find that the mean volumes of respiration for normals and schizophrenics are 7·98 and 7·68 litres respectively, while the mean effective volumes of respiration are 5·43 and 4·58 litres respectively. The effective ventilation of schizophrenic women, therefore, is only 84 per cent. of that of normals, whereas the total ventilation is 96 per cent. of normals. These figures again show that the shallow rapid method of respiration is less efficient than the deep slow method.

Melancholics.—Nineteen female melancholics were found to have an area of body surface which was smaller than that of normal females (Table I).

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* $D = \frac{E(a - e)}{a - i}$ where D = volume of dead-space in c.c.

E = volume of tidal air in c.c.; e = percentage of CO_2 in the expired air (average 4·1); a = percentage of CO_2 in alveolar air (average 6·0); and i = percentage of CO_2 in inspired air (average 0·04).

The smaller size, however, was barely significant. The volume of tidal air was smaller than that of normal females, but the difference (27 $\pm$ 14·70 c.c.) was also not significant.

Regarding their rate of respiration, for 19 female melancholics this was 19·55 $\pm$ 0·685 as against 18·61 $\pm$ 0·494 for normal females. This difference was not significant.

It would appear, therefore, that the difference in volume and rate of respiration in schizophrenics does not apply to melancholics. The number of male melancholics (six) was too small to give significant results. These on the average breathed more deeply and rapidly than normals (Table I).

Comparison of Schizophrenics with Normal Subjects in a Dozing and Sleeping State.—In the last two lines figures are given showing the respiratory depth and rate of normal women first in a dozing and secondly in a sleeping state, in order to show the difference from schizophrenics. In these experiments the subject was lying in a chest made on the same principle as the box described above. I am indebted to Dr. Antonovitch for these records. It will be seen that in the dozing state the breathing is slow and deep, but the effective ventilation per minute is about the same as for schizophrenics, 4·60 litres per minute as against 4·64. In sleep the depth is greatly diminished, being rather smaller than that of schizophrenics (317 as against 339 c.c.); the rate becomes slightly faster than in the dozing state, and the total effective ventilation is about half of that of normal subjects.

DISCUSSION

The fact which stands out most clearly from the foregoing results is that schizophrenic patients on the average breathe more shallowly and more rapidly than normal subjects. Melancholic patients do not show the same type of breathing, but resemble normals.

Inefficiency of Schizophrenics' Type of Respiration.—It has been shown that where the total ventilation is the same the shallow rapid type is less efficient than the deep slow type. It does not follow from this, however, that the oxygen consumption is necessarily less in these subjects than in normals. This can only be ascertained by direct measurement, and further work on this subject remains to be done. At present we can only suspect that the inefficient action of the respiratory bellows might lead to deficient oxygen consumption.

With regard to patients in a more severe catatonic state, Gullotta [4] has remarked that catatonics breathe more slowly than other schizophrenics. My own observations support this view. It may be, therefore, that the shallow breathing of schizophrenics is ordinarily compensated for by the increased rapidity of respiration, but that when the rate falls during an increase in the catatonic state then failure of oxidation ensues, with cyanosed extremities and increased torpor of mind. The question as to how far this condition is

dependent on deficient oxygen consumption could best be solved by giving these patients pure oxygen and seeing whether their state improved. So far, certain facts indicate that increase of breathing from administration of CO_2 or from forced exercise (Peters [8]) improves the condition. It is possibly this improvement of respiration which explains why there are fewer catatonics to be seen in those mental hospitals where the patients are encouraged to take a relatively large amount of exercise.

Effect of Type of Respiration on Reaction to CO_2 Inhalation.—The fact found by Golla and co-workers that schizophrenics failed to respond with increased ventilation so quickly or to the same extent as normal subjects may perhaps best be explained by supposing a depression of the respiratory centre. It is possible, however, that the schizophrenic by virtue of the shallowness of his breathing does not allow the CO_2 to accumulate so quickly in the alveoli, and that therefore the respiratory centre is not affected so quickly.

Comparison of Type of Respiration in Schizophrenics with that in Sleep.—One rather paradoxical fact has emerged, namely, that the type of respiration is the exact opposite of that found in individuals who are in the drowsy state between sleeping and waking. A German writer [9] has made a thorough study of the resemblances of the condition to schizophrenia. Yet the breathing in the drowsy state is deep and slow. It is when the drowsiness is replaced by sleep that the respiration takes on the same shallow character as in schizophrenics; in sleep, however, it remains much slower.

Bearing of Type of Respiration on Susceptibility to Tuberculosis.—The type of respiration in schizophrenics is of interest in another connexion. As is well known, tuberculosis is particularly frequent among these patients. This has often been attributed to their poor physique generally. The present study, however, suggests that the shallow character of their respiration might render the lungs more liable to the disease, owing to their insufficient expansion.

SUMMARY

1. Sixty-two normal subjects and 121 schizophrenics were examined as to the depth and rate of their respiration by means of a plethysmographic apparatus. The schizophrenics were found to breathe more shallowly and more rapidly than the normals, the difference being significant. This difference did not apply to 25 melancholic patients.

2. It was pointed out that the type of respiration employed by the schizophrenics was an inefficient one. The significance of this different type is discussed in relation to oxygen consumption, and to its possible bearing on the catatonic state with its stupor and cyanosed extremities; also in connexion with the relation of schizophrenics to tuberculosis, and to the dozing and sleeping states.

This work was done during the tenure of a Commonwealth Research Fellowship at the Maudsley Hospital, London. The collation and elaboration

of results were worked out during the tenure of a Pinsent-Darwin Studentship from Cambridge University.

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CEPHALIC DYSOSTOSIS

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INTRODUCTION

It is not unusual in neurology and psychiatry to discover that signs which have at first been considered minor assume on further investigation paramount importance. Nice examples of this are found in the Argyll Robertson pupil and the extensor plantar reflex. It is possible therefore that those inconsiderable variations in structure which have been labelled 'stigmata of degeneration' may with the growth of knowledge at least repay us for the trouble of investigation. The importance acquired by acromegaly which was first regarded as a curiosity should emphasize this fact. The association of epilepsy and amentia—both rather mysterious conditions—with variations in cephalic structure compels us to regard these anomalies as the result of disease-processes capable of being understood.

A number of morbid states, some sporadic and some familial, have been described, all of which are characterised by anomalous development of the skull. Some of these have been associated with somatic variations elsewhere. It is these disease-conditions which it is proposed to classify together under the title of 'Cephalic Dysostosis,' and it is hoped to show that they may depend on a common pathogeny. The morbid conditions which form this group are oxycephaly, acrocephalosyndactyly of Apert, the hypertelorism of Greig, the craniofacial dysostosis of Crouzon, scaphocephaly, trigonocephaly and plagiocephaly. Microcephaly, arachnodactyly, and cranio-cleido-dysostosis are not included, since it will be seen when an attempt is made to define the characteristics of this group that these affections are not admissible.

The conditions classified here as cephalic dysostosis are supposed to result from premature synostosis of the skull bones. This ætiology has never been subjected to criticism, which is curious, since disease of the soft parts is a frequent concomitant. The contention of this paper is that the affections concerned are the result of temporary retardation or even cessation of the growth of the brain, which allows premature synostosis and consequently distortion of the skull when the brain resumes growth.

RÉSUMÉ OF DISEASE-CONDITIONS CONCERNED

A short description will be given and the theory of their ætiology will be described.

Oxycephaly was first described by von Graefe [1] in 1866. It is characterized by a high tower-like skull, exophthalmos, strabismus (usually divergent)

and nystagmus. The supraorbital ridges, glabella and frontal eminences of the skull are obliterated. The increased intracranial pressure is frequently shown on skiagrams by digitations and clinically by severe headaches and optic atrophy. The association of oxycephaly and syndactyly is fairly common and has been named by Apert [2] *acrocephalosyndactyly*.

Hypertelorism is a rare state which was described by Greig [3] in 1924. It is characterized by excessive width between the eyes, a retroussé nose with a broad bridge and widely open nasal passages, strabismus (usually divergent) and prognathism.

Craniofacial dysostosis was first described by Crouzon [4] in 1912. It is characterized by a central frontal boss, a beak-like nose, prognathism, exophthalmos and strabismus. *Scaphocephaly* is produced by a narrow skull with a sagittal ridge. *Trigonocephaly* is excessive scaphocephaly producing a triangular skull. *Plagiocephaly* is asymmetry of the skull caused by unequal growth of one of the lateral halves. This produces an unequal, lopsided appearance.

Cases have been described of all these conditions (except perhaps plagiocephaly) in which they occur in familial and hereditary incidence. Craniofacial dysostosis is according to Crouzon practically always hereditary, but Comby (quoted by Crouzon) records a solitary case. Oxycephaly is usually described as occurring sporadically, but cases have been recorded in which it has been inherited in certain families. A few familial cases of scaphocephaly have been reported. Any of these states appear to be capable of producing signs of increased intracranial pressure varying from the mild headaches in Crouzon's cases to the severe headaches with vomiting occurring in oxycephaly. Epilepsy occurs in some. A large number of patients with hypertelorism, scaphocephaly or trigonocephaly suffer from epilepsy. It is tempting to attribute the epilepsy and the amentia which so frequently accompanies this group to the increased intracranial pressure, but this is not justified. It is in oxycephaly that the intracranial pressure is highest and yet oxycephalics are frequently of normal mentality. In hypertelorism the intracranial pressure is not necessarily much raised above the normal, yet in this condition fits and amentia are common. It is necessary therefore to appreciate that the morbid process is not confined to the bones or sutures, as has generally been affirmed.

The incidence of amentia in these cases can be correlated with the tendency to epilepsy, since in craniofacial dysostosis (where the mentality is usually normal) epilepsy is rare, and it did not appear in Crouzon's cases except as infantile convulsions. Conversely, in hypertelorism epilepsy and amentia are both common.

Anyone who has examined skiagrams of these conditions must feel convinced that they belong to a common group. Moreover, the fact that they are occasionally intermixed (as in Chotzen's [5] case, which showed acrocephalosyndactyly, hypertelorism, and dysostosis craniofacialis; in Roubino-

vitch's case—quoted by Crouzon—of craniofacial dysostosis with trigonocephaly, and Lesne's [6] case of craniofacial dysostosis and oxycephaly combined) suggests that one disease-process may be at the root of these curious anomalies. The morbid process affects now one part of the skull and now another; hence the variations produced appear to be different states, although in reality they are only manifestations of one pathology.

PATHOGENESIS

In the past it has been the custom to attribute such affections to 'degeneration.' Many of these patients, however, are of normal mentality. Other writers have been dissatisfied with the vague word 'degenerate' and have evoked the process of 'blastophoria' as though this would save further intellectual and clinical effort. It is true that gross poisoning with lead and other toxins or that violence to the ovum may lead to the production of monsters, but it is very doubtful whether the embryo is ever subjected to such a severe environment during intrauterine life. Moreover, the published cases of hypertelorism contain only one instance in which a parent was alcoholic or otherwise diseased. The presence of parental disease in oxycephaly, acrocephalosyndactyly, or craniofacial dysostosis is also rare. Before one can accept the blastophoric origin of these conditions much more evidence must be produced.

Jansen [7] would argue that the cause is the pressure of the amnion upon the fœtus and even gives a table suggesting the anomalies produced by applying pressure at various dates. For instance, application of pressure in the second and third week produces anencephaly, in the fourth to the fifth week acrocephalosyndactyly, in the seventh week mongolian idiocy, and so on. This theory is a mass of assumptions and may be dismissed as not only unproved but very unlikely to be correct. In comparison with acromegaly and cretinism it is tempting to seek a glandular basis, but although Crouzon described a slight goitre in his first case of craniofacial dysostosis none of his other cases showed any glandular disease. The cases of hypertelorism which have been described have usually been in children who have not appeared to be suffering from glandular troubles. We cannot therefore accept a frank endocrine basis for these conditions such as we find in acromegaly.

Crouzon explains the premature fusion of sutures in craniofacial dysostosis by Mendelism. He thinks that the disease is a recessive and explains its rarity in this way. However, it appears to occur occasionally as a mutation and breeds as a dominant. Oxycephaly is usually explained as being due to 'inflammation' causing premature synostosis. No signs of this 'inflammation' have ever been seen nor any reason for its occurrence been produced. Moreover, 'inflammation' is associated with hyperæmia, whereas synostosis usually occurs with diminished blood supply—for example, in old age.

Greig pointed out that the reason for the widely separated eyes in his cases appeared to be the development of that part of the sphenoid which is laid down in cartilage, so that the lesser wings grow as large as or larger than the greater wings. Greig, Reuben and Fox,[8] Ogilvie and Pozel [9] all point out that scaphocephaly is due to underdevelopment of the great wings of the sphenoid. In oxycephaly the greater wings are enlarged. No explanation is produced for these curious variations in sphenoidal growth.

Crouzon believes that craniofacial dysostosis is due to the premature fusion of certain sutures (he does not state exactly which ones), and that the subsequent deformities are caused by an effort to accommodate the growing brain. He points out that the patients with the most deformed skulls suffer least from increased intracranial pressure. He divides the process into three stages: (1) stage of premature fusion of the sutures; (2) stage of deformity to accommodate the brain; (3) stage of increased intracranial pressure. It will be seen that he regards the disease as being basically one of the bony parts —the sutures.

PERSONAL THEORY

I believe that these explanations are incomplete mainly because they are based on the study of the bones to the neglect of the brain.

It has been admitted since the failure of operative measures in microcephaly that the growth of the skull is dependent on the growth of the brain. In microcephaly the brain does not grow to the usual size and the skull shapes itself to conform to it. On the contrary, in macrocephaly the skull grows large to accommodate the larger brain. Again, in porencephaly the skull is often flattened over the undeveloped half of the brain. It will therefore appear probable that any deformity of the skull is the result of cephalic disease and not of disease of the bones or sutures. For some unknown reason the fœtal brain does not develop at the normal rate and in the normal directions. Possibly the brain even stops growing for a time. This uneven development allows the sutures to fuse where they are in contact. Possibly the normal stimulus for the junction of the sutures is the cessation of stimuli from the growing brain when it has reached an appropriate size. It is true, as Greig says, that in oxycephaly ' where the bones touch they fuse,' but the bones should not come into contact, and if the brain is growing normally they have no time to fuse. In cephalic dysostosis the brain, as it were, hesitates and is lost.

We are now in a position to understand the various cranial anomalies which have been considered hitherto as different morbid states.

Whether oxycephaly, hypertelorism, craniofacial dysostosis, trigonocephaly or any other member of the group is produced will depend on which part of the brain is sluggish in its growth. If it is mainly the frontal lobes which are affected, so that the coronal sutures are allowed to fuse (possibly other sutures also fuse), the brain will find itself forced to enlarge the skull upwards, producing oxycephaly. If the parietal lobes are at fault then

scaphocephaly is produced. Hypertelorism and craniofacial dysostosis appear dependent on disproportionate growth of the occipital and possibly other lobes (this may explain the abrupt occiput in hypertelorism).

It is in plagiocephaly that this explanation is shown most clearly. Here the skull is underdeveloped on one side so that it is markedly asymmetrical. The writer believes that this is due to temporary cessation of growth of the whole of the telencephalon on the affected side. This is demonstrated by the fact that in porencephaly (where the whole telencephalon on one side may be replaced by a cyst) there is a similar skull defect on the affected side. Moreover, if the unilateral defect were caused by premature fusion of one coronal suture one would expect that the growing lateral lobe would cause displacement of the third ventricle, such as one finds in cerebral tumours of one cerebral lobe. That this displacement does not occur is shown in Jacobsen's [10] case. Unfortunately no reliable series of plagiocephalic brains has been examined, so that the question cannot be finally settled on this evidence. It is a justifiable question to demand why the brain should stop growing and then start again while the child is still in utero. There seem two possible explanations. (1) Either the mother or child is born with a poor glandular equipment which tends to become exhausted and then recover at some period during parturition. (2) Some toxic influence interferes with the cerebral growth of the embryo.

The glandular theory is not necessarily incompatible with Mendelian views and is upheld by one remarkable fact. In anencephaly where the skull does not grow—possibly because there is no cephalic stimulus—there are anomalous adrenal bodies. This was first noticed by Morgagni in 1723, who recorded that the adrenal glands in anencephalics were small but not absent. In 1910 Elliot and Armour [11] discovered that this was due to the absence of fœtal adrenal cortex so that the adrenals of anencephalics approximate to the adult type. Boyd [12] has since confirmed this and the fact may be considered to be well established. The fœtal brain is apparently dependent on some product of the adrenal bodies (unless it is vice versâ).

The toxic theory cannot be dismissed without consideration. No little evidence can be produced in its favour. For instance, oxycephaly is a fairly frequent concomitant of hæmolytic jaundice occurring in children (Feingold and Case,[13] Mikulowski [14]). It is possible that this produces a toxic encephalitis or affects the brain by poisoning the glands primarily.

Bronfenbrenner [15] records a case in which a woman was forced to leave Alaska because it was feared that the lack of fresh food would affect her pregnancy. She left Alaska in the later months of gestation and eventually gave birth to a child suffering from acrocephalosyndactyly. Whether this was due to toxic conditions or lack of vitamines it is difficult to say.

The syndactyly which occurs in Apert's disease is frequently put forward as proof that this condition is one of the bones. It seems probable that the fusion of the fingers and toes may be due to deficient innervation of these

parts. It will be noted that the arms and legs do not fuse but only the distal parts, which need more delicate innervation to move them. Moreover, it is in oxycephaly—a condition in which it is postulated that the frontal lobes are affected and which would lead to loss of spontaneous movement—that fusion occurs. It appears probable that the fingers and toes are normally prevented from fusion in early intrauterine life by their spontaneous movement; otherwise it would seem that pressure would interfere with the blood supply and would inevitably cause fusion in such a closely packed organism. Any condition of the brain leading to immobility of the fingers and toes would lead to their fusion before birth and may be the explanation of the occasional occurrence of these deformities in cases of simple amentia. This fact is upheld by the cases in which the fingers and toes are fused only by their soft parts—for example, the toes in Bostock's [16] and Johnstone's [17] cases. This in my opinion makes it difficult to believe that the disease is one of bony origin. Poor innervation with loss of spontaneous movement during intrauterine life may explain the occurrence of genu valgum (such as appears in the case recorded below) and similar deformities of the elbow, which Apert calls 'acrocephalosynankie.'

The myopia which is a frequent concomitant of cephalic dysostosis must be regarded as another proof that the disease is one of the soft parts and not of bony origin. Whether the myopia is present at infancy or has any connexion with the aberrant growth of the nervous tissue or the increased intracranial pressure is uncertain. The anosmia which sometimes occurs may be the result of cerebral disease, the increased intracranial pressure, or pressure from the distorted skull, but which is the causal factor it is difficult to say.

We may define cephalic dysostosis as a distortion of the skull caused by increased intracranial pressure due to abnormal growth of the brain allowing premature synostosis. That there is a Mendelian factor present appears probable, but the object of this paper is to suggest that the primary disease is one of the brain rather than the bone. The Mendelian theory is not incompatible with the glandular or toxic theories, but indeed in some cases no Mendelian factors are discoverable. The case recorded below occurred in such a large family of siblings that even a recessive had ample opportunity of appearing more than once.

It is admitted that we have carried the investigation of the pathogeny only one step further back, yet the advantages of this theory of causation are obvious. It brings the whole rather chaotic collection of facial and cranial deformities into one orderly classification and offers one explanation for their occurrence. Moreover, it suggests prophylactic measures which theories of fundamental bony disease have caused to be regarded with apathy and despair.

PERSONAL CASE

This case is one of hypertelorism occurring in a woman of 58 and accompanied by amentia, epilepsy, and a paranoid psychosis.

Family History.—I am informed by the patient's sister that family albums—now unfortunately no longer extant—showed the patient's ancestors on both her mother's and her father's side back to her great-grandfathers. No abnormality is said to have been present in these people.

The patient's father married at 23, and by this wife had the following normal children.

1. William. He died at the age of 60 with a cerebral tumour.
2. Rose. She is still alive, aged 75, with 10 children, eight of whom are alive.
3. Charlotte. She was crippled through a fall while pregnant. She died aged 56.
4. Millicent. Still alive, aged 70. She has three children. One died from (?) spinal caries. The other two are alive, married, with healthy children.

FIG. 1.—Note the widely separated eyes and contour of the skull.

5. James. Died aged 18 from phthisis.
6. Frederick. Alive, aged 65, one son married.

The patient's father's first wife died at the age of 40 and he married again, a woman aged 23. She was not consanguineous. The children of this union were :

1. Edith. The patient.
2. Lousa. Alive, aged 54. No children.
3. George. Alive, aged 53, single.
4. Charles. Alive, aged 51. Two children, one a girl who is married, with one healthy child.
5. Violet. Alive, aged 50, with six children—one son is married, but with no children.
6. Henry. He died aged six months from vaccinia.

These children were followed by three premature births. The cause of this is unknown. None of these people—other than the patient—has suffered from hypertelorism, amentia, or insanity as far as can be ascertained.

Personal History.—The patient was a full-time child. The labour was protracted and instruments were used. She was noticed to have peculiar facial conformation after birth. She is said to have been a bright happy little child and got on well at school although she did not reach higher than the sixth standard when she left at the age of 14. At the age of 17 she started to suffer from petit mal attacks which were thought to be fainting fits and no medical advice was sought on their account. Her husband describes how these attacks would occur in the midst of some domestic duty such as laying the table or arranging crockery on the dresser. After the momentary interruption she continued her occupation where it had been left off.

She married at the age of 32 and gave birth to a normal child two years later. She then started to suffer from grand mal attacks. These appeared at the menstrual

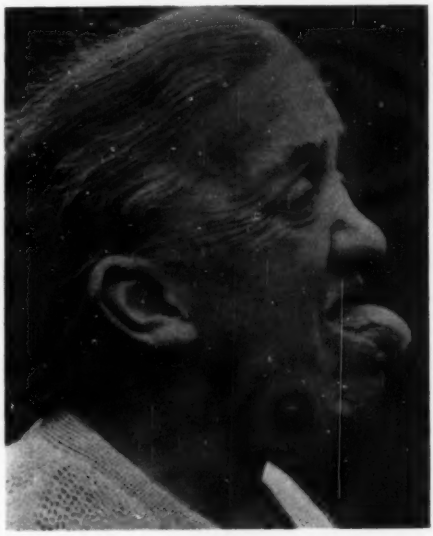

FIG. 2.—Note the humped-up tongue caused by the prognathism.

periods and were usually nocturnal. They were preceded by an aura of suffocation. She did not consider these attacks sufficiently severe to merit treatment.

Six years ago she fell downstairs in a fit and was badly concussed. As she is said to have been unconscious for two and a half days it is probable that some cerebral damage—possibly vascular—was present. When she recovered consciousness she was noticed to behave abnormally. Her sister states that 'when she came round she behaved like a mad woman. She talked in a nasty sharp way and pulled you about. She would say to me, " I don't want you—go home." ' She grew worse for three years, becoming more and more hostile and persecuted. She became hallucinated and screamed for no apparent reason. In 1930 when she became violent the family were forced to send her to the local infirmary where she was disorientated, impulsively violent, and noisy. She stated that she could hear her sister calling to her. She was certified and sent to Bexley.

On admission she was noted to be disorientated, confused and resistive. She stated that people were discussing her and kept putting out her light. She felt that

there were plots against her and was very complaining, querulous, and dissatisfied. Every day she complained of physical violence and of attempts to 'do her in.' She was described as being degenerate in appearance, but was not recognized as suffering from hypertelorism.

She recovered after being in Bexley for approximately two months, having little memory of the acute phase of her illness. She had two fits while in Bexley, but no note was made of their type.

She remained comparatively well until September 1933, when she complained of hallucinations of smell and taste, thought that her bed was haunted and that ammonia was being sprinkled on her. She became agitated and depressed, and was again certified and sent to Bexley.

She was admitted by the writer who recognized her as a case of hypertelorism, but on admission she was too noisy and violent to allow a proper physical examination.

FIG. 3.—Note shape of skull.

She suspected that the physical examination was an attempt at sexual and physical assault and shouted 'You're not going to murder me, are you?' She kept shrieking that she had been examined by a black doctor, that patients were being blinded in hospital for their money, and that everything stank of ammonia.

She appeared in rather a Korsakovian state of disorientation with confabulation two days after, and stated that she had been for a motor ride. She was still suspicious and said to me, 'You're not going to petrify me, are you?' Since this she has passed through a slight euphoric stage hardly amounting to hypomania, a stage of confusion and quiet contented stages. Since admission she has had occasional fits—about four per month—but these are now controlled by luminal.

Physical Condition.—She is a rather stocky woman, 4 ft. 11½ in. in height. Her cranial condition is well shown in the accompanying photographs. She has a rather scaphoid, dolichocephalic head without the abrupt occiput commonly associated with hypertelorism. The top of the skull is slightly triangulated anteroposteriorly and laterally. The eyes are widely spaced with the palpebral fissures sloping somewhat downwards laterally. There is some ptosis and some exophthalmos. The nose shows

the characteristically wide base with a flattened bridge and broad tip. The nasal passages are large and the nasolabial folds deeply marked. The mouth is fairly normal, but the lips are thin. The mandible projects and the teeth are prognathic, irregularly spaced, and do not meet those of the upper jaw. The ears are not a good shape, but would pass for normal on a normal head. She has no other somatic abnormality except for genu valgum of the right knee. It is interesting to note that Montford's [18] case of hypertelorism showed a similar deformity.

Mr. P. M. Moffatt, the consulting ophthalmologist, kindly examined her eyes and reported on them. He found a divergent strabismus, and slight nystagmus which is

FIG. 4.—Note the shape of the skull, the ptosis, strabismus and slant of the eyes. The broad bridge of the nose is well shown, and the humped-up shape of the tongue on protrusion.

not always present. The right fundus showed a blurred margin to the disc and narrowing of the vessels. There was a patch of choroidoretinal atrophy below and a smaller similar patch above the disc, also a little scattered fine choroidoretinitis which was thought to be of old standing. 'Vitreous floaters' were present and slight early peripheral lens striæ. The left fundus showed similar changes but not so marked. Myopia was found to be present in each eye, about 4 D., but she was able to read only 5/60 distance. She could read Jaeger 3 with each eye without glasses. The movement of the eyes was found to be normal, but her convergence was poor—possibly because of her exophthalmos. She had some bilateral ptosis. Her eyes reacted to light and accommodation.

The other cranial nerves were examined and her sense of smell was found to be very poor. She said that oil of lavender smelled ' very strong like ammonia,' and that asafœtida smelled of vinegar.

The fifth nerve is normal and the sense of taste appears normal. The face moves normally and hearing is good. Ninth, tenth, eleventh, and twelfth nerves show no abnormality.

No abnormality was discovered in the rest of the nervous system.

The cerebrospinal fluid was taken and the Wassermann and Meinicke reactions found to be normal. Unfortunately the pressure was not taken. I have tried since to do so but was prevented by a dry tap.

FIG. 5.—Anteroposterior X-ray picture of skull.

The other systems are normal. Her blood pressure is 120/80, which is low for her age.

Intelligence Tests.—She did not co-operate well and was inclined to let her attention wander. She was more successful in concrete than in abstract tests.

Binet Simon ... ... M.A. 8·4/12.
Burt's Graded Reasoning test ... ... M.A. 7.
Porteus tests were unsuccessful owing to her poor sight.
She reads fairly well and has some idea of what she has read.

Measurements.—The measurements of her skull are of interest.

Longest anteroposterior dimension ... ... 26·6 cm.
Greatest breadth ... ... 14·3 cm.
Cephalic index ... ... 70.
The skull is therefore dolichocephalic.
Interpupillary distance ... ... 8·3 cm.
Normal = 6·0.

Distance between inner canthi	4·0 cm.
Distance between outer canthi	11·3 cm.
Orbital height	4·0 cm.
Orbital width	4·2 cm.
Ear to ear over glabella	32·0 cm.
Ear to ear over bregma	31·5 cm.
Ear to ear over lambda	31·0 cm.
Nasion to inion	39·5 cm.

Weight of patient, 8 st. 9½ lb. Height, 4 ft. 11¼ in.

The *skiagrams* are of much interest. The lateral view shows the greatly elongated dolichocephalic appearance one would expect. The frontal bone is somewhat abrupt and the anterior end of the head is rather square, but true oxycephaly is not present.

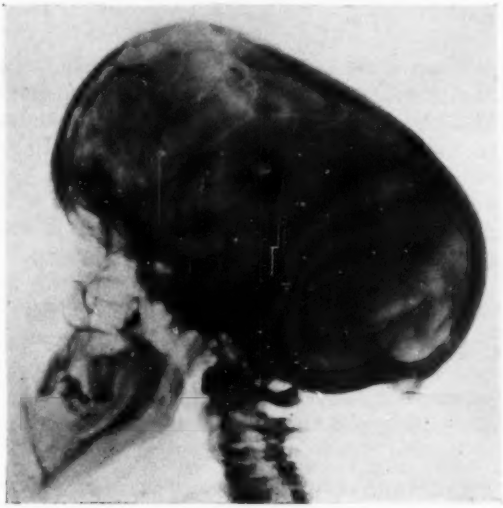

FIG. 6.—Lateral X-ray picture of skull.

The occiput is not flattened as it usually is in hypertelorism. The bone over the posterior end of the skull is thick and solidly formed. There is a curious appearance over the point of junction of the frontal and parietal bones. There is an area of rarefaction, or of separation of the external and internal tables of the skull, which almost looks as though an air sinus had been inserted there. This appears to be the vestigial remains of the anterior fontanelle. The base of the skull is so distorted that it is difficult to be sure if the sella is abnormal. There is evidence of old increased intracranial pressure in the stippled 'beaten-silver' appearance of the skull. The prognathism and the large irregular teeth are well seen. There appears to be slight distortion of the upper vertebræ, probably due to the unbalanced cumbersome skull.

The anteroposterior view is very dark because of the immense length of the skull. The widely separated orbits are well seen. The large irregular teeth show up well in the upper and lower jaws.

The patient has a daughter who is apparently normal both in appearance and mentality. She was born after a normal labour and was breast-fed. There is nothing

of interest in this girl's childhood. After she left school she worked as a milliner but married eight years ago and gave birth to a normal child six years ago. This child—a boy—appears normal and is quite bright. I have had no opportunity of applying mental tests or examining physically either the child or the mother.

This is the first case of hypertelorism recorded in the literature in which the condition has been associated with schizophrenia or any other psychosis. It is doubtful if these conditions are related; psychological factors appear to influence her psychotic state, if they are not causal. Her hostility to her sister is apparently founded on the fact that her husband courted her sister at the same time that he paid attention to her. She makes no mention of her facial condition in her psychotic episodes although it is known she is rather sensitive about it.

The writer wishes to thank Dr. Clark, Medical Superintendent of Bexley Mental Hospital, and the London County Council for permission to utilize the case published above, also Mr. Sands, pharmacist to the Hospital, for the trouble he has taken in photographing the patient and printing the skiagrams.

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Short Notes and Clinical Cases

'CROWDING' OF INHIBITION AND OF EXCITATION

By

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In a former paper [4] on the pathogenesis of narcolepsy I alluded, on p. 9, to five cases, reported by Kinnier Wilson,[8] Cave [1] and Rosenthal,[7] in which the patient had made the observation that attempting to fight off a sleep attack sometimes gave rise to an attack of powerlessness. For this phenomenon I proposed an explanation. Sleep, as we know from the work of Pavlov, arises through the irradiation and coalescence of many scattered inhibitory processes. When a person fights sleep off, a multitude of excitatory processes come into play, opposing the irradiation of the inhibitory processes. The latter, when not entirely checked, continue to irradiate where they are least opposed. They may invade the motor areas, if these areas are (as in narcolepsy they presumably are) unduly 'inhibitable.' When this happens, the patient will find that in attempting to prevent sleep he has caused an attack of powerlessness to supervene. I suggested that this phenomenon may be likened to what happens in a game of American football, when the player carrying the ball is 'crowded' by his opponents, so that he runs in the direction in which he sees the least opposition. Thus we may say that in the five cases mentioned above the warding off of an impending sleep attack caused the inhibition then gathering in the substrate of consciousness to be diverted or crowded into the motility substrate.

A cognate phenomenon occurred in a case reported by Cohen,[2] which I cited on the same page. In this case the patient gave the following account of one of her cataplectic attacks: 'I think I could have stopped it in the first stages, but unfortunately I was stupid enough to concentrate not on sitting still, but on trying not to spill the coffee from the cup I was holding, with the result that I kept the coffee cup quite safe, but slipped to the floor beside it.' This is a particularly clear illustration of the crowding of inhibition from one part of the brain to another—in this instance from the central mechanism of the upper limb to those of the trunk and the lower limbs.

In the present paper I shall consider the question: Are there cases in which one may assume that there has been crowding of excitation? I believe that this question may be answered affirmatively. The clearest

instances are those in which a person voluntarily suppresses an impulse to some movement. I shall cite several instances under three headings.

1. *Instances in which there is Suppression of a Primitive Motor Response.*— I refer to a significant observation made by MacCurdy [6] in regard to the reactions of soldiers to their first bombardment. The reader should consult the paragraph beginning at the foot of p. 16 of MacCurdy's article (this paragraph is cited in full on p. 125 of my more recent paper [5]). After remarking that the commonest response is fear, MacCurdy said: 'A less common reaction is that of excitement, accompanied even with a kind of spurious elation. The man has a tendency to make facetious remarks about the shells, to laugh at feeble witticisms, and very often feels under considerable *motor tension, there being a pressing desire to do something, to do it immediately and do it hard*' (italics mine). I suggest that the motor tension observed by MacCurdy may be regarded as a manifestation of crowding of excitation. The 'most basic' wish of the front-line soldier is to run away to a spot where there are no shells.* To say that the impulse to run away is suppressed is to say that excitation is crowded away from the nervous arrangements utilized in running. This excitation cannot vanish into nothingness, and its persisting in the motility substrate may be inferred from the fact that the soldier is aware of motor tension and of 'a pressing desire to do something, to do it immediately and do it hard.'

It may be presumed that MacCurdy's soldiers experienced tension in the muscles of the lower limbs as well as in the other muscles of the body. Therefore, we may not conclude simply that excitation was crowded from lower limb areas to areas in which the lower limbs are least represented. The crowding must have occurred not in the middle, but in the highest motor centres. In the highest centres there is a multitude of 'nervous arrangements' in which the lower limbs are prominently represented. It therefore seems possible, in the highest centres, for excitation to be crowded in such a way that the lower limbs are tense, though the impulse to run away has been suppressed.

There is possibly a second reason for the tension experienced by the soldiers. Man's natural way of fighting calls into play the large muscles. To be moved into a front-line trench must therefore 'awaken' powerful motor impulses. But modern warfare is anomalous in the fact that the front-line soldier, instead of fighting, as nature intended, with his large muscles, spends most of his time in a trench not really fighting at all or, at most, fighting with his index finger. It is possible that, when the soldier is required to discard primitive in favour of more 'refined' methods of fighting, excitation,

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* I say the 'most basic' rather than the 'strongest' wish. In most soldiers the wish to appear brave is stronger than the wish to run away. But the wish to run away is ontogenetically the older of the two. The soldier who actually runs away is acting more automatically than he who suppresses the impulse.

crowded away from the nervous arrangements utilized in hand-to-hand fighting, is diverted into other parts of the motility substrate, a subjective feeling of tension being one of the consequences.

2. *Instances in which there is Suppression of Speech.*—Speech being a special form of movement, the instances I am about to give are identical in principle with those given under the previous heading. Every observant person knows that many people, when telling a lie, betray themselves by making accessory movements, e.g., wiggling a foot.* These accessory movements frequently help one to determine whether one is listening to the truth. Furthermore, there are instances in which, without telling a lie, one must suppress something one wants to say. For example, a debater, hearing his opponent's argument, immediately thinks of the answer he wants to make, but, of course, must remain silent until his turn comes to speak; he may react by restlessly drumming with his fingers.

3. *Instances in which there is Suppression of both Speech and More Primitive Movements.*—The best example here is that of the angry man who would like to assail his opponent both physically and verbally. The suppression of these movements may mean the crowding of excitation into nervous arrangements representing other movements; the frustrated man may slam the door or, at his first opportunity, kick the cat.

It should be emphasized that crowding of excitation does not account for *all* the phenomena that occur in consequence of the suppression of strong impulses. When one attempts to suppress an impulse, there is, in the centre in question, a sort of tug of war between excitatory and inhibitory processes. The resulting phenomena must therefore be carefully separated into two groups: (*a*) those arising from the crowding of excitation into *some* neighbouring centres, as discussed in this paper, and (*b*) those arising from an overflow of inhibition into *other* neighbouring centres. The latter group of phenomena were discussed on p. 11 of my earlier paper,[4] with particular reference to several cases in which sleep and other manifestations of inhibition occurred in consequence of the suppression of the defence reflex to pain and of sexual reflexes. In these instances one sees the manifestations of an excess of inhibition. In the paragraph beginning on p. 16 of MacCurdy's article, to which I have already alluded, the existence of these two groups is easily seen. MacCurdy found that some soldiers, at their first bombardment, reacted with motor tension, and with a 'pressing desire to do something, to do it immediately and do it hard'; here is crowding of excitation. Other soldiers, in the same situation, reacted with pathological sleep; here is overflow of inhibition.

It is possible that in some cases one may see, at one and the same time, manifestations resulting from crowding of excitation and those resulting from overflow of inhibition. I once studied a bright thirteen-year-old boy,

* Hohman [3] called particular attention to this phenomenon.

referred for disobedience and other 'behaviour problems.' His mother, an intelligent person, said that whenever he did something wrong his reaction to his attempt to keep it secret was to become very talkative. We deal here with a boy who wished to tell his mother certain things and at the same time wished not to tell her. In other words, the nervous arrangements for uttering certain propositions formed the scene of a tug of war between excitation and inhibition. Since the boy refrained from talking about his escapade, but jabbered away about other matters, we may infer that excitation was crowded from the nervous arrangements representing one set of propositions to those representing other sets. At the same time, it is possible that in addition to this crowding of excitation there was also some overflow of inhibition throughout the highest layers of the highest centres, resulting in loss of control over lower layers which then became overactive, overtalkativeness being the consequence. If this be true, his overtalkativeness was the result of two factors which reinforced each other, in very much the same way as contraction of the biceps and relaxation of the triceps reinforce each other in producing flexion at the elbow.

SUMMARY

When by voluntary effort one succeeds in preventing inhibition from supervening in a given area of the brain, this inhibition may be 'crowded' into a nearby area. For example, several narcolepties have reported that when they fight off an impending sleep attack, an attack of powerlessness sometimes supervenes. Corresponding to this crowding of inhibition, there occur, in other cases, phenomena attributable to crowding of excitation. For example, MacCurdy observed that during bombardment some soldiers experienced great motor tension. Since in front-line soldiers the impulse to run away has been suppressed, it seems reasonable to suppose that the motor tension observed in many of them occurs as the result of the crowding of excitation from one part of the motility substrate into some of its remaining parts. Other examples are given.

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A NOTE ON CUTANEOUS AND MENINGEAL HÆMANGIECTATIC NÆVI ASSOCIATED WITH CONTRALATERAL EPILEPSY AND/OR HEMIPLEGIA (SYNDROME NEURO-CUTANÉ) *

By

A. J. WILSON, Coventry

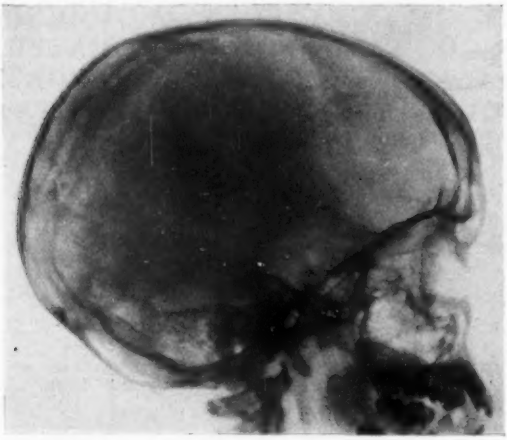
PERSONAL CASE

The patient is a girl, age 10, an only child, who was brought to hospital with the complaint of 'attacks during which she becomes paralysed all down the right side.'

The history is that the birth was normal, and the birthmark on the left side of the face was more vivid then than now. She was quite healthy until the age of six months,

* This case formed the subject of a communication at the meeting of the Association of British Neurological Surgeons at the National Hospital, Queen Square, London, on December 8, 1933.

when she had a generalized epileptic attack involving the whole body. No paralysis was noticed, but the attacks recurred until she was 12 months old and then ceased.



In the intervals between she was quite normal. She walked at 12 months, and weakness of the right side was then first noticed, the arm being held limp from the elbow and the leg dragged slightly. With massage the paresis gradually improved.

At the age of two years, the present Jacksonian attacks commenced, but she has

- - - - - 1° red.
——— 1° white.

been able to attend school regularly and has been quite active. The child knows when the attacks are coming ; she says ' I'm bad, mama.' Then jerking of the right side of the body sets in, commencing in the face and spreading to the right limbs. She does not lose consciousness, knows her mother and can reply to questions, though in a

mumble. The turns last only a few seconds, and she sleeps afterwards and is generally limp and quiet for a few days. The hemiparesis is accentuated during this time—she cannot pick anything up and drags the leg in walking—but it then gradually improves. The frequency of the fits is about once every 3–4 weeks. In the intervals between, she is quite well. The maternal grandmother had fits, but there is no history of skin nævus.

The patient is a bright intelligent child of normal physical development. There is a 'port-wine stain' nævus involving chiefly the skin area of the first and second divisions of the left trigeminal nerve, the inner margin ending quite sharply in the midline. The right limbs are shorter, smaller and weaker than the left. She is a left-handed child. There is no noticeable abnormality in the gait. There is weakness of the right side of the face, but the remaining cranial nerves are normal. Deep reflexes in the right limbs are all increased; the right plantar response is extensor, and the right abdominal reflexes are weak.

Ophthalmic Report.—Vision, full. Pupil reactions—direct, normal. Wernicke's hemianopic reaction not present. Fundi and retinal vessels normal. Slight conjunctival injection of left eye, of a type allied to the facial nævus. No vascular abnormality of left iris on slit-lamp examination. No buphthalmos in left eye. Tension of both eyes normal (right 12, left 15). Fields of vision show a well-defined homonymous hemianopia with loss of the right half of each field. No hemianopic scotomata.

X-ray of Skull.—Left half smaller than right. A sinuous calcified shadow visible in occipital region above the level of the tentorium, extending upwards and forwards to central region of hemisphere: (?) a similar shadow visible also in anteroposterior view.

Wassermann negative. Remaining systems normal.

DISCUSSION

Most of the recorded cases of the angiomatous malformations of the brain have been unexpectedly met with at exploratory craniotomy for Jacksonian epilepsy, hemiplegia or tumour and have not been associated with cutaneous nævus, and in consequence there was nothing to help in the preoperative pathological diagnosis. All the series described by Cushing in his book, *Blood-vessel Tumours of the Brain*, and Sargent's six cases were of this type. On the other hand, in a minority of cases of intracranial angiomatosis it has been possible to make such preoperative pathological diagnosis from the presence of cutaneous and especially facial nævus in the patient, an association which has long been known and has recently been described in a few cases as familial. In neither of the groups has the histopathology of the brain lesion been carefully worked out, but it seems to be established that when intracranial lesions are to be expected in a case of cutaneous nævus they will be found to be of the group of angiomatous malformations and not of that of the true tumour hæmangioblastomata. Even here, however, an occasional exception has been recorded (quoted by Lindau) in which two cases of cerebellar hæmangioblastomata occurred in association with skin nævus (type of latter not stated). The angiomatous malformation in such cases with cutaneous nævus is usually of the venous type, although Soutter has met with one of arterial origin. As, however, according to Cushing, venous angiomata tend later in their course to become increasingly aneurysmal, with secondary

arterialisation of the veins, arteriovenous angiomata may be expected to be found occasionally in the patients of more adult years.

Such lesions are said to be always situated above the tentorium. However, cases of both venous and arterial angiomata have been found in the hindbrain, although in these instances no skin nævus was present. The venous malformations may be simple varices or may be widespread racemose congeries of vessels, many of which extend far beyond their focal localization, and while apparently superficial, really penetrate deeply into the brain in a wedge-shaped prolongation, base to the surface and apex terminating near the ventricle, into which a terminal hæmorrhage may occur. This type would be better termed ' cerebromeningeal angioma.' The process may extend also on to the mesial and basal surfaces of the hemisphere. In certain cases—more especially those associated with cutaneous nævus—calcification may occur either in the walls of the vessels, in their thrombosed channels, or in the membranes surrounding them, and may then become visible in an X-ray film. In such instances it may be possible to distinguish the deep penetrating racemose type, in which surgical treatment is out of the question, from the truly superficial variety, where, however, the operative results have been very little better owing to the great vascularity not only of the pial angioma but also of the dura and bone. In some of the cases of the latter type in which operation has been performed, an external hæmorrhage has occurred later through the dura and trephine aperture with the production of a large hæmatoma, as in Bathe Rawling's case, in which the epilepsy was relieved by the operation. Similar angiomata may occur in connection with the spinal meninges, associated in isolated instances with skin nævus of the corresponding metameric surface.

The cutaneous lesion present in this syndrome may be the ordinary ' port-wine stain ' or nævus flammeus, or it may also have a more deep-seated cavernous component, when actual hypertrophy of the bones or limb may be seen ; it may be pigmented or it may be a nævus anæmicus. The process may involve the buccal or nasal mucous membranes. It may be sharply demarcated to the trigeminal skin area, even more sharply than in zoster, and when it is, according to Cushing's cases, the associated intracranial (meningeal) nævus may topographically correspond, so that in a nævus involving the first trigeminal skin area the corresponding portion of the dura innervated by that branch may be chiefly involved. But it may not be confined to the face, and very extensive skin areas of the entire body may be involved, but in that event one side of the body or face may be predominantly covered ; the intracranial nævus is then to be expected on that side. In three cases, however, the meningeal angioma was found at operation or post mortem to be contralateral to the facial cutaneous lesion. In another typical instance with skin nævus and contralateral epilepsy, at autopsy no intracranial vascular malformation was found. Grieg's patient, explored for focal epilepsy, with an angioma venosum extending into the

cortex found at operation, had adenoma sebaceum of the face, a condition which he states to be a cutaneous tumour of bloodvessel origin.

Associated with the cutaneous nævus may be present certain ocular abnormalities in the eye of the affected side. There may be buphthalmos from congenital glaucoma with enlargement of the pupil. Abnormal vascularity may be found in the iris by slit-lamp examination. The retinal veins may be unduly tortuous, the retinal background may be a darker red, while externally the sclera may have a bluish ' china-doll eye ' appearance. In a few cases these ocular findings have been associated with intracranial angioma but without any skin nævus. Capillary hæmangioblastoma of the retina (v. Hippel's disease) is not found accompanying cutaneous or intracranial angioma, but is instead a part of Lindau's syndrome, where similar cystic hæmangioblastomata are found usually in the cerebellum, though in Barnard's case it was in the forebrain.

Visual defects of peripheral origin do not occur in the venous angiomata as, at first at any rate, the lesion is not an expanding one, though if by pathological anastomosis it becomes transformed into an arteriovenous (aneurysmal) angioma, then increased intracranial pressure and papillœdema follow. Visual defect of cortical origin has occurred with the present case, in four other instances, and in every one a facial nævus was found and the intracranial lesion shown to be calcified by X-ray examination.

Lastly, mental defects varying from irritability of temper to idiocy have been a marked feature of many of the cases, as is instanced by the number reported from asylums, especially in Brushfield and Wyatt's series. Such mental changes have been ascribed to a gliosis of the cortex or to hæmorrhage from the intracranial nævus, but another possibility seems to be lack of cerebral development, as the X-ray films of many show the affected side to be considerably smaller than its fellow. The latter may also be the explanation of the hemiplegia or epilepsy.

Editorial

MENINGEAL HÆMORRHAGE

IN the nature of things it does not appear likely that meningeal hæmorrhage can ever alter its aspects and in some obscure way become different from what it always has been. As regards its analogue, cerebral hæmorrhage, with which indeed it is very often found in unison, no conspicuous change has come over our conceptions of its pathogenesis and types, although no doubt knowledge of its mechanism in some particular circumstances has of late been amplified. But in respect of the other an increasing number of case-reports are being published entitled 'Subarachnoid hæmorrhage,' with or without the epithet 'spontaneous' to qualify it. Older expressions such as 'meningeal apoplexy' are losing or have lost their vogue, and the idea would seem to be gaining ground that the subarachnoid variety is a disease-condition per se, with clinical features of its own, independent of other states, and recognisable at sight, so to speak, by the recovery of blood from the cerebrospinal fluid. No doubt the chief, possibly the sole, reason for this is the ease with which lumbar puncture is performed and with which effusion within the theca can be detected. Yet this facility is apt to prove misleading, for it imparts a spurious simplicity to the subject, one which in reality it is far from possessing.

Another possible explanation of the rise into prominence of this topic is related to the occurrence of so-called congenital aneurysms on cerebral vessels and to their disclosing themselves by rupture and extravasation of blood through the subarachnoid system. That these lesions appear to develop with greater frequency than was formerly supposed is not merely a plausible surmise, but receives support from actual examination. For instance, among 124 cases of subarachnoid hæmorrhage collated by Symonds in 1924 a ruptured aneurysm was discovered in only 11, but since that time the percentage has risen curiously; no less than 15 of 16 cases autopsied by Biemond and ter Braak (1933) were verified as having been thus produced. The natural consequence seems to be that a

stage has been reached where bleeding under the arachnoid, of sudden onset perhaps, though not invariably, and particularly when it occurs in persons under the age of 50, is assigned to congenital aneurysm without a thought of any other interpretation. But the facts militate strongly against so artless a view. Whereas no signs of hypertension or arteriosclerosis were observed by the Dutch neurologists mentioned above in their series of cases, exactly the opposite was noted by Strauss and his colleagues (1932), who in every one of 11 cases of subarachnoid hæmorrhage confirmed postmortem—though the average age of their patients was no higher, if not indeed actually lower, than that of the subjects examined by Biemond and ter Braak—found signs of either local or generalized arteriosclerosis, with aneurysmal formation in seven of the series. In other words, the lesion is attributed by the former to congenital aneurysm, by the latter to arteriopathic disease of a different kind altogether. Here is a clinical and pathological conflict which cannot easily be explained away.

Concentration of attention on the subarachnoid variety of meningeal apoplexy and on congenital aneurysm has been conducted at the expense of pathological principles and clinical observation. When blood is found in the spinal fluid the mere fact provides no clue to its provenance. Blood can come from arteries, veins, capillaries, and sinuses ; from any of these it may leak, and in different directions. From without inward meningeal hæmorrhage might be schematically classified as extradural, dural, subdural, subarachnoid, pial, and subpial respectively ; while not all are of equal importance or frequency, some are likely to be combined. To what extent any may occur strictly by itself is a point on which knowledge is vague, and sorely in need of statistical amplification. The subarachnoid variety, like all meningeal effusions, is merely a complication of some other lesion. Blood within the membranes is nothing else than an incident in a process whose seat may be remote and character diffuse. Subdural hæmorrhage often bursts through the fragile arachnoid, which may be torn, too, by blood originally extravasated underneath it. Further, bleeding may start under the pia and escape into the arachnoid system. To diagnose subarachnoid hæmorrhage, therefore, because of the presence of blood in puncture fluid, is in a superficial sense correct, but in no other ; it is to mistake a sign for the lesion, a symptom for the process, a termination for a cause. And even when the condition is diagnosed by the spinal fluid, of itself it can furnish no information as to the underlying

morbid state. *Pace* the simplifiers of the day, it tells us exactly nothing in respect of the latter. The symptoms and signs of intracranial aneurysm are not those of its rupture, which may or may not take place. And numerous other lesions are liable to be overlooked when aneurysm is complacently regarded as the sole cause of the condition. For example, syphilis and a variety of other infections, different kinds of meningitis, alcoholism, heatstroke, sinus occlusion, and still other states are all likely enough to induce subarachnoid bleeding. The first sign of an otherwise symptomless cerebral tumour may be escape of blood. Of no little interest, too, is the apparent association of migrainous attacks with subarachnoid effusion. To ascribe these last-mentioned symptoms to congenital aneurysm, as is often done to-day, encounters many obstacles; it is hard to understand how the process should continue for years, sometimes many years, before culminating in hæmorrhage, and equally so to account for the migrainous type induced if, as is customary, the aneurysmal lesion is at the base. When not verified postmortem the inference that aneurysm has been the cause of both 'migraine' and subarachnoid effusion may be altogether mistaken.

Claims, however, have been advanced to exclude the foregoing causes of subarachnoid hæmorrhage by alleging that the type for which the term should be used and to which it should be restricted can be distinguished as being at once 'spontaneous' and 'massive.' Both considerations are erroneous. The former adjective is at times employed to-day as though it were identical with or equivalent to 'causeless,' which is absurd. Though extrinsic factors may seemingly be lacking, intrinsic factors can not; the condition of the vessel wall, which cannot be foretold, governs the situation; the coats give way not 'spontaneously' but because they are compelled to do so. If synonymous with 'non-traumatic' the question of what constitutes 'trauma' has first to be answered. As for the other qualifying term, it cannot be defined; who shall say, and on what criterion shall he rely in asserting, that an effusion is 'massive'? Aside from this crux, every degree and grade of effusion take place even in aneurysm, if we are to believe those who affirm that aneurysms leak and give rise to no more than headache and giddiness at one time, but inundate the arachnoid system at another.

The usefulness and clinical value of the subarachnoid conception, then, would seem to be seriously impaired if it is made too exclusive, and if it is regarded as standing for a

morbid entity. But if the form is put in its proper place, as a manifestation of a process of varying causation, and if it is realized to be often nothing more than a part of meningeal and cerebral hæmorrhage in general, it becomes a matter of genuine significance, less a thing-in-itself than a clue to what has been going on within the skull.

Abstracts

Neurology

NEUROPATHOLOGY

[155] **The pathological anatomy of myasthenia gravis** (Zur pathologischen Anatomie der Myasthenia).—M. N. Zajewloschin. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1933, **148**, 28.

A woman developed symptoms of typical myasthenia gravis when she was 30 years of age and death occurred after one year of illness. At autopsy a tumour was found in the anterior mediastinum just over the pericardium. The microscopical appearance of the growth resembled that of the thymus. The thymus itself was in the normal position and the growth at a slightly lower level, unlike the cases described by Weigert and Hart, in which a tumour of similar type replaced the thymus. The tumour is best termed a non-malignant 'thymus growth.' In the thyroid gland accumulations of lymphocytes were found between the follicles. The pituitary was enlarged but showed no microscopical changes. The suprarenal glands were enlarged, the cells of the zona fasciculata contained excess of lipoid and in other parts of the cortex much pigmentation could be seen. In the ovaries many cysts were present, while the bloodvessels were enlarged and surrounded by lymphocytic infiltration. The thymus was slightly enlarged.

In the skeletal muscles many accumulations of lymphocytes were found, but muscle-fibres were unaltered. Similarly, the nervous system showed considerable lymphocytic infiltration in the hypothalamic region. In addition, nerve-cells were partly degenerated, especially in the sympathetic ganglia. Thymus tumours, lymphocytic infiltration of the muscles and alterations in the nervous system have been described in myasthenia gravis, but it is rare to find all these changes in a single case.

M.

[156] **The influence of the sympathetic nervous system in the ætiology of Erb's progressive muscular dystrophy** (Zur Frage der sympathicogenen Entstehung der Erb'schen Muskeldystrophie).—H. Curschmann. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1933, **143**, 335.

Ken Kuré has reported alterations in the sympathetic nervous system in several cases of progressive muscular dystrophy; in animals he found that

dystrophic changes in the thigh muscles follow the extirpation of the sympathetic chain. Curschmann has investigated 26 cases of Graves' disease in which the cervical sympathetic had previously been removed. His object was to ascertain if such extirpation was the cause of muscular dystrophy, but in one case only of the 26 was a slight alteration in the muscle condition apparent. The results, therefore, do not favour Kuré's theory.

M.

[157] **Functional circulatory disturbances and epilepsy** (Funktionelle Kreislaufstörungen und Epilepsie).—W. SPIELMEYER. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1933, **148**, 285.

SOME years ago the author affirmed that the alterations in the cornu Ammonis of epileptic subjects are the consequence of functional disturbances of the circulation. Researches carried out later by him and his co-workers have led him to the conclusion that alterations found in the cerebellum, the brain cortex and not infrequently in the cardiac tissue of persons subject to epilepsy are also the consequences of the same process.

M.

[158] **Studies in epilepsy.**—F. L. MCLAUGHLIN. *Jour. of Ment. Sci.*, 1933, **79**, 688.

THE acid-base equilibrium of the blood in epilepsy is here studied, measurements being made of pH, alkali reserve and lactic acid. The results suggest that in the interval period between fits variations in alkali reserve and lactic acid are usually within normal limits, but those of pH extend to a range slightly above normal. The average lactic acid concentration is normal. The average alkali reserve and pH values are slightly above normal. The averages of results obtained from alkali reserve and pH during the two-hour period preceding the fit show no significant difference from those of the interval period. Results obtained from blood samples of individual patients before, during and after a particular seizure are illustrated by graphs, and lead to the following conclusions: (*a*) there is a fall in alkali reserve during and for varying periods after the fit, this fall being accounted for by the accumulation of lactic acid in the blood. Decrease in lactic acid during recovery is accompanied by increase in alkali reserve. (*b*) The blood pH falls during the fit, due firstly to apnœa during the tonic stage, and secondly to production of lactic acid during the clonic stage. Recovery to the normal pH is more rapid than in the case with normal individuals after exercise.

C. S. R.

[159] **The factor of hypoglycæmia in the ætiology of idiopathic epilepsy.**—R. L. Haviland Minchin. *Jour. of Ment. Sci.*, 1933, **79**, 659.

Epilepsy is associated with a low fasting blood sugar. The glucose tolerance curve in this disorder demonstrates the islets of Langerhans to be overactive. Those drugs which are beneficial in epilepsy raise the blood sugar level (except bromides, which reduce the irritability of the cerebral cortex). In the post-convulsive phase of epilepsy immunity from fits is associated with a raised blood sugar. Variations in the balance of the autonomic system have little influence on the incidence of convulsions. Natural recovery from epilepsy is associated with the onset of hypoinsulinism.

C. S. R.

[160] **The presence of gastric acidity in multiple sclerosis.**—O. H. Perry Pepper and George Wilson. *Amer. Jour. Med. Sci.*, 1933, **186**, 773.

This communication gives the results of an investigation of the gastric acidity in 11 cases of disseminated sclerosis. The study was promoted by the alleged analogy between this disease and Addison's anæmia with subacute combined degeneration of the spinal cord, and also by the fact that the authors met with one case of undoubted disseminated sclerosis which showed achlorhydria without any complication likely to cause such anacidity. The usual Ewald test meal was employed and the graphs obtained from fractional gastric analysis are given in each case.

Free hydrochloric acid was present in all cases and in fair amounts, the original case which suggested the investigation being the only one in which free hydrochloric acid was absent.

C. W. D.

SENSORIMOTOR NEUROLOGY

[161] **A special form of multiple sclerosis related to Friedreich's ataxia** (Ueber eine besondere, der Friedreich'schen Tabes nahestehende Form familiärer Sclerosis multiplex).—B. Brouwer. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1933, **148**, 321.

Two sisters had suffered for many years with symptoms suggesting a diagnosis of Friedreich's ataxia. One of these patients died and histological examination of the central nervous system showed, in addition to the manifestations of the combined sclerosis found in that disease, plaques similar to those of disseminated sclerosis. The author discusses the question whether this case is a special form of Friedreich's ataxia or of disseminated sclerosis and is more

in favour of the latter view. Referring to the nature of disseminated sclerosis, he considers that endogenous causes are of greater importance than generally supposed.

M.

[162] **Non-familial spasmodic paraplegia of Strümpell type** (Paraplégie spasmodique du type Strümpell non-familiale).—L. VAN BOGAERT. *Jour. de Neurol. et de Psychiat.*, 1933, **33**, 453.

A CHILD, age eight years, showed a slowly developing spasmodic paraplegia. There were no sphincter troubles and no cerebellar symptoms. The fundus oculi was normal. Two lumbar punctures showed a permeable subarachnoid space and some increase in the protein content of the cerebrospinal fluid. The paraplegia was accompanied by pronounced cervicodorsal kyphosis. According to the author this combination of symptoms is characteristic. Several other members of the family were examined with uniformly negative results. In the same generation as the patient, however, gastric and duodenal ulcers, verified surgically, occurred in three of five maternal collaterals, and in the paternal stock there was a predominant asthenic tendency.

C. W. D.

[163] **A special form of meralgia paræsthetica—nothalgia paræsthetica** (Ueber paraesthetische Neuralgien und eine besondere Form derselben: Nothalgia Paræsthetica).—M. ASTWAZATUROW. *Deut. Zeits. f. Nervenheilk.*, 1934, **133**, 188.

MERALGIA paræsthetica (Bernhardt) has been well known for many years. In this affection of the external cutaneous nerve of the leg the patient suffers more from paræsthesia than from pain. A short time ago Wartenberg described a similar affection in the superficial branch of the radial nerve. The author of this paper has seen the same disturbance in other cutaneous branches of the radial (musculospiral) nerve and in the posterior divisions of the spinal nerves. He believes that the radial nerve, the posterior spinal nerves and the external cutaneous nerve of the leg are a special group which are more liable to paræsthesia than to pain. In the case of the fifth cranial, the median, and the external popliteal nerves, also the lateral branches of the spinal nerves, painful sensations are more liable to occur.

M.

[164] **Relation of jugular foramina to epileptic seizures and hydrocephalus.**—D. B. DAVIS. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 351.

THE author has devised a technique for the measurement of the jugular foramen by means of X-ray photography. Of studies made on 40 patients

with epileptic convulsions 29, or 72·5 per cent., had foramina which were larger on the right, and 11, or 27·5 per cent., on the left. Ten cases with varied diagnoses were studied, and nine were found to have the larger foramen on the right and one on the left.

The combined areas of the two foramina in the convulsive state group of 10 cases were found to be less in seven instances than the group presenting varied diagnoses.

Seven cases of hydrocephalus were X-rayed, but in six observations the foramina were too minute to permit accurate measurement.

R. G. G.

[165] **Thrombosis of a superior cerebral vein.**—D. B. Davis. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 22.

Only two cases of this syndrome have previously been described in the literature. The present case was that of a thrombosis of the left rolandic vein, probably due to venous stasis, in a man suffering from an acute infection and a low blood-pressure with some measure of sclerotic change in the vessels. The following neurological signs were present: semi-stupor; paralysis of right lower face, arm and leg; a bilateral Babinski sign; a complete right-sided hyperæsthesia, loss of ability to recognize objects placed in right hand, and inability to determine texture with the right hand. An exploratory operation was performed, but the patient died shortly after.

R. G. G.

[166] **Hemihypertonia postapoplectica.**—M. Molitch. *Jour. Nerv. Ment. Dis.*, 1932, **76**, 25.

In three cases described by von Bechterew of apoplexy occurring before the tenth year of life with sudden onset and varying degrees of unconsciousness, the clinical picture was distinguished by an absence of paralysis and the occurrence of tonic spasms on the affected side. This condition should be clearly separated from other forms of posthemiplegia. The lesion is probably located in the striopallidal system.

R. G. G.

PROGNOSIS AND TREATMENT

[167] **Therapeutic results of air injections in the spinal canal in epilepsy** (Über therapeutische Erfahrungen mit der lumbalen Lufteinblasung bei epileptischen Anfällen).—R. Friedmann and T. Scheinker. *Deut. Zeits. f. Nervenheilk.*, 1933, **133**, 35.

The authors have treated 43 cases of epilepsy including the so-called genuine, symptomatic, traumatic and senile forms with injections of air into the

subarachnoid space through the lumbar portion of the spinal canal. In 25 per cent. of cases the result was good, in 28·3 per cent. a partially good result, while 36·6 per cent. were unchanged. In 10 per cent. the result could not be affirmed. The authors state they have never seen permanent damage in consequence of this treatment.

M.

[168] **Ketogenic diet in epilepsy.**—S. M. ALLEN. *Jour. of Ment. Sci.*, 1933, **79**, 677.

THIS investigation was on 20 cases of idiopathic epilepsy of various types over a period of 16 months. Under the influence of this special diet 13 had a reduction in fits, two were quite unchanged, and five had an increase. It would seem that it is during the night, in the period when the patient is farthest from his meal, that any increase in fits generally takes place (during ketosis). Most epileptics would welcome a regime that changed their time of fits from day to night. Therefore this result of the treatment can also be considered satisfactory. The writer concludes that even in institutional epileptics of the lowest grade, ketosis engendered by a high fat diet is antagonistic to seizures in a fair number of patients. The results may be more favourable in children. There are difficulties in maintaining the diet, but in well-regulated hospitals it should not be so. The reason why ketosis should limit the number of convulsions is not easy to discover. If the establishment of acidosis is of value in treatment, it follows that the administration of alkali should result in an increase of seizures. In some patients, although acidosis is actually present, continued giving of alkali results in fits. This suggests that it is the sudden change from acidosis to alkalinity that precipitates them. Hyperpnœa in epileptics frequently brings about the precipitation of a fit. In rapid deep expiratory breathing the patient gets rid of CO_2 from the blood, with the result that the alkalinity increases; a seizure is the result. If, however, the patient is made to re-breathe the air he has actually expired the expected seizure does not occur. It would appear that oxygen lack in the tissues is a fundamental condition. A decrease in CO_2 in blood results in a bias towards alkalinity, therefore acidosis is a condition in which the tissues much more readily receive oxygen, and alkalosis a condition in which the tissues find difficulty in consuming oxygen. In epilepsy the cells most affected are the neurones, and it is well known that in oxygen deficiency the neurone system is the first to suffer. The suggestion is not made that epilepsy is a syndrome depending on an abnormal alkaline bias in the blood and tissues. This may be the cause, but oxygen supply to the tissues can be affected in many ways besides decreasing CO_2 in the blood. The blood supply may be at fault, as in spasm of arterioles, or there may be stasis of the venous return. The essential cause of epilepsy remains unknown. It is perhaps nearly related to angina pectoris, migraine, and general muscle

cramps. All these conditions are alike in that there may be many precipitating factors quite different from the essential cause.

C. S. R.

[169] **Modern treatment of myopathy with glycocol** (Le traitement moderne des myopathies avec le glycocolle).—M. SCHACHTER. *Gaz. des hôp.*, 1933, **106**, 1385.

AMERICAN observers have succeeded in demonstrating that glycocol plays a prominent part in the physiology of muscle and that on its presence depends the formation of creatin. In myopathy there is a disorder of creatin metabolism. On these facts, the authors of this paper base their treatment by glycocol (glycine) in muscular dystrophies. Under the influence of 15 grammes of glycocol per day, primary myopathies are said to benefit, whereas other forms are not influenced. The first sign of improvement in those cases treated was indicated by a sensation of tingling, of contraction, then of tremor in the affected muscles; next, fatiguability tended to disappear and finally movements improved. It was noted that several months might elapse before improvement was manifest and that it was not always equal in the different groups of muscles. Treatment by glycocol should be started early as those cases in whom muscle fibres are still intact react best to the treatment. This explains why better results are obtained in children. In certain cases, one may have to fall back on an associated form of treatment, e.g. with hepatic glycocol and testicular glycocol.

C. W. D.

[170] **Treatment of neurosyphilis with acetarsone (stovarsol) given intravenously.**—L. SPIEGEL. *Amer. Jour. Syph. and Neurol.*, 1934, **18**, 56.

RECENT chemical research has succeeded in purifying acetarsone, the compound '594' of Ehrlich and Hata, so that it can now be used safely for either subcutaneous or intravenous injection. For the latter purpose the sodium salt is prepared by dissolving 1 gm. of the drug in 9 c.c. of normal sodium hydroxide and after solution adding 11 c.c. of sterile distilled water. The dosage recommended is 0·25 to 0·5 gm. for the first three or four doses, rising up to 1 gm., injections being given once or twice a week for extended periods. Sézary has recommended a total dosage of 21 gm. for each course of treatment.

Greater powers of penetration, due to a more crystalline structure, are claimed for pentavalent compounds of arsenic such as tryparsamide and acetarsone than for the more colloidal trivalent compounds. The author has found that acetarsone, unlike tryparsamide, has no tendency to affect the optic nerves, and that it has produced no untoward effects in the 25 patients whom he has treated.

J. G. G.

[171] **The physiology of electropyrexia.**—C. A. Neymann and S. L. Osborne. *Amer. Jour. Syph. and Neurol.*, 1934, **18**, 28.

It has recently been shown that the treponema pallidum is killed in vitro by heating to 42° C. for one hour, or to 41° C. for two hours. Bessemans and Thiry have shown that this also holds good for human infection. The present authors have therefore tested the body temperature in various organs by means of thermo-couple needles placed deep in the tissues during treatment by diathermy, radiothermy and electric blankets. The sites chosen for insertion of the needles were the liver, Hunter's canal, the lumbar theca and the cisterna magna. In addition rectal and axillary pyrometers were used. It was found that the rectal temperature was raised nearly a degree higher than that within the theca, which approximated more closely to the axillary temperature. The authors found it possible to keep the internal temperature of the body, including the cisterna magna and lumbar theca, as high as 42° C. (107·6° F.) for periods of two, or in some cases four, hours.

In treating primary syphilis by this method there was no difficulty in healing the sore, but owing to the early dissemination of spirochætes secondary rashes were apt to appear in parts, such as the face and scalp, in which the temperature had not been raised to the necessary height.

J. G. G.

[172] **Improvement in nervous and mental states under cortin therapy.**—F. A. Hartman, G. M. Beck and G. W. Thorn. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 1.

Cortin, a special preparation of the adrenal cortex, was administered first in a group of cases suffering from Addison's disease and later in other conditions in which there was evidence of nervous and muscular fatigue. It was found that cortin therapy gave a varying degree of relief in the nervous and mental symptoms in the cases reported. These comprise three cases of Addison's disease, three showing fatigue without demonstrable adrenal insufficiency, and five organic neurological cases. Two cases of myasthenia gravis were treated but showed no improvement.

R. G. G.

Psychopathology

PSYCHOLOGY

[173] **Writing slips and personality.**—A. A. Roback. *Character and Personality*, 1933, **1**, 137.

The purpose of this investigation was to study the conscious experiences as well as the objective phenomena resulting from interference between graphic impulses, and also if possible to formulate from the results a neural theory

explaining the various phenomena. The author made a study of all the *lapsus calami* and typographical errors reported in Freudian literature, and was surprised in the first place at the paucity of the material and, secondly, at the naïveté of the interpretation, the uncritical attitude toward a scientific problem. To begin with, not all misprints are really unintentional, though the majority of misprints are involuntary, and in a few cases it can be granted that some complex is at work. Perhaps the term ' complex ' is too strong for this mechanism, because the cause and maybe even the excrescent element have been in the consciousness of the individual some time previous to the occurrence. In a review of psychoanalytic interpretations a simpler cause with no exception was found in the graphic mechanism of the writer as affected by the concatenation of the characters in the context and the mental tension at the time. Fatigue will favour repetition of previous characters, while emotional excitement, hurry, will tend to favour anticipation. That the lapses are largely of an unconscious origin the writer is convinced. Morton Prince suggests the possibility of a co-conscious image directing the hand. The processes takes place in the unconscious. In assimilation and composite formations remarkable adjustments occur, combinations, compromises, etc. The unconscious is the locus of a series of fairly complicated physiological processes in a dynamic setting, but not displaying the motivated traces of past experiences as the Freudians would have it. Our general handwriting is probably not free from unconscious motives, but lapses, because of their nature, their suddenness or capriciousness, lack the coherence and integration of the more *incubated* unconscious, the unconscious which lies at the root of motivation. For an unconscious wish or complex to counteract the effect of an ingrained habit, it would have to become conscious, and even then to contest the goal of the dominant determining tendency in the motor trait. When we look to the physiological explanation in at least the vast majority of writing lapses, there is no need for treating experimentally induced slips differently from those made in ordinary life. On the Freudian view one is obliged to invoke two different methods of approach. In ordinary lapses, the *meaning* is to be examined in the light of past emotional experiences, while in experimentally induced mistakes only the *mechanical* context—letter formation—can help ; for the same word or symbol in one place throws up one kind of slip, while in another, in conjunction with another word or symbol, either before or after it, it will reveal a slip of another kind.

C. S. R.

PSYCHOSES

[174] **Schizophrenia and neurosyphilis** (Schizophrenia et neurosyphilis).—G. I. Odobesco and H. Vaselesco. *Ann. méd.-psychol.*, 1933, **91**, 652.

The authors have collated various views as to the possible relationship between schizophrenia and neurosyphilis, particularly dementia paralytica.

Schizophrenia and dementia paralytica may appear separately or together in individuals suffering from neurosyphilis. If schizophrenia appears alone the question arises, (1) whether the syphilis is responsible for the manifestation, or (2) the precipitating factor which lowers the threshold to the incidence of the schizophrenia, or (3) whether there has occurred a chance combination of two syndromes in the same individual each having no relationship to the other.

It appears that if the patient with the characteristic blood and cerebrospinal fluid changes of dementia paralytica and suffering from schizoid manifestations is subjected to malarial therapy, the syphilitic condition may regress to a simple neurosyphilis without evidence of cortical involvement, but the schizophrenia continues unchanged. The evidence with reference to this finding is, however, contradictory. The prognosis in the schizoid form of dementia paralytica the authors regard as definitely bad.

They conclude by quoting three cases of schizophrenia-neurosyphilis all of which received antisyphilitic treatment but no malarial therapy. One patient died and his brain showed the typical lesions of dementia paralytica. One, who gave a history of recurrent attacks of schizoid trouble which had occurred long before the development of neurosyphilis, seemed to recover and relapse spontaneously in a fashion quite independent of the neurosyphilis or its treatment. In the third case the schizoid phenomenon appeared at the same time as the neurosyphilitic manifestations : the patient improved while receiving specific treatment. The authors conclude that in our present state of inadequate knowledge and without statistical evidence at our disposal it is unwise to attempt to deduce the nature of the relationship between these two conditions.

C. W. D.

[175] **A fasting blood-sugar in schizophrenia.**—W. FREEMAN. *Amer. Jour. Med. Sci.*, 1933, **186**, 261.

A SERIES of 59 male schizophrenic patients were studied as regards the levels of the blood-sugars. Six samples were taken at regular intervals. In 95 per cent. of 347 determinations the figure lay between the limits of normality, viz. 80 and 100 mg. per cent. The average level was 86·6 mg. as compared with an average of 95·4 mg. in 31 normal control subjects. Eight readings were obtained in the hypoglycæmic and eight in the hyperglycæmic ranges. The variation among the schizophrenics was higher than in the controls as shown by the coefficients of variation, 11 and 6·9 per cent. respectively.

The samples were collected in pairs at 15-day intervals with intervening periods of two and a half months. There was a consistent drop in the average blood-sugar level from period to period, the first being 99·3 mg. and the third 93·1 mg. It is suggested that repetition may lead to a reduction of emotional

reaction to the test and that this plays a part in determining the blood-sugar level. The range of difference in consecutive tests also decreases consistently from one period to another; in the first test the average difference was 10·6 mg. and in the last 8 mg. per cent. No correlation was established between the blood-sugar level and the age, duration of stay in hospital or the severity of the disease. Hebephrenic cases showed the highest average level, viz. 99·7 mg., and katatonic cases the least, viz. 92·7 mg. The author concludes that schizophrenia is characterized by normal fasting blood-sugar levels, but that the individual variation is greater than in normal subjects.

C. W. D.

[176] **Manic-depressive 'exhaustion' deaths.**—Irving M. Derby. *Psychiatric Quarterly*, 1933, **7**, 436.

Of the 386 deaths of manic-depressive patients during the past five years at Brooklyn State Hospital, 48 per cent. had accompanying exhaustion and excitement. An analysis of this fraction, representing 187 deaths, showed that over 20 per cent. were wrongly diagnosed. The remaining 148 histories were investigated for relative findings that might contribute to an understanding of the large number of 'exhaustion' deaths. There was a ratio of 3 to 1 of manic to mixed types, with a scattered few in the other types. One-fifteenth of the number were male deaths. Ninety per cent. died before the fifth decade. A history of improper nourishment and loss of sleep occurred in more than a third. Also contributory to a physical exhaustion preceding admission were childbirth (12 per cent.), febrile disease (6 per cent.), and severe methods aimed at weight reduction (3 per cent.). One hundred and sixteen (almost 80 per cent.) died within two weeks of admission, and 10 per cent. within the first 48 hours. Prolonged treatment in tepid pack and injudicious use of continuous baths were contributory death causes. Improper nourishment and failure to utilize subdermal infusions early also promoted a fatal termination. Of 20 autopsies of death from exhaustion, in eight signs of toxic-infection were present; there was acute cardiac dilatation in 8 per cent.

C. S. R.

[177] **Regression in manic-depressive reactions.**—August E. Witzel. *Psychiatric Quarterly*, 1933, **7**, 386.

As a result of his studies the writer concludes that there is a regression of the individual's thinking, feeling and behaviour in this state to levels of development which are older. This regression varies in the individual case, and can only be measured by a more or less complete knowledge of the psycho-

logical history. The general goal of regression differs—negation of life-death, in the depressed, and, to begin life anew, in the manic. These opposed reactions may occur in the same individual during the course of an attack, or one may appear in one attack, and the other in a subsequent one. In many cases there is evidence of failure to outgrow the instinctual phases of both the anal and oral organization, leading to a turning away or retreat from interests in or relationships to objects outside the individual, to self as the object—to a state of narcissism. What is assumed to be deterioration is more apparent than real, and no patient should be considered hopeless therapeutically because of apparent profound regression. The presence of marked preoccupation with somatic complaints and the free use of the projection mechanism must lead to a guarded prognosis. Suicidal attempts per se are not necessarily of bad omen. Many, under the age of 20, seem to regress rapidly because probably they have not had time or opportunity to develop defence or compromise-formation and sublimations. Advancing age appears to be conducive to diminished object libido and may between the ages of 50 and 60 seem to make little effort to progress. The lack of incentive to become well is more marked at this age period.

C. S. R.

PSYCHOPATHOLOGY

[178] **Mental disorders in pernicious anæmia** (Ueber Geistesstörungen bei perniciöser Anämie).—D. FRANCKE. *Allg. Zeits. f. Psychiat.*, 1932, **98**, 93.

THREE cases of pernicious anæmia with mental symptoms are described. In the first case the mental picture is that of a chronic paranoid psychosis, and in the second case a subacute paranoid psychosis with disorientation and suicidal impulses. The main feature in the last case was a general depression of all mental functions followed by a terminal dementia. In two of the cases the blood picture was considerably improved by liver therapy, but there was no parallel improvement in the mental symptoms.

In all three subsequent autopsy and pathological examination were carried out. This demonstrated a considerable degree of brain-cell alteration, especially shrinkage and disintegration ; there was also some liquefaction of cells. In the blood vessels hyaline degeneration of the middle coat and areas of fatty degeneration in the intima with an overgrowth of endothelium were noted. From the pathological findings, no direct relationship between the changes in the blood-vessels and the cellular alterations in the brain could be traced. It is not surprising that the author supports the theory of toxic action on the nervous system in pernicious anæmia rather than the view that the anæmia itself is responsible for the pathological changes.

L. Z.

[179] **The inheritance of mental defect.**—R. Ruggles Gates. *Brit. Jour. Med. Psychol.*, 1933, **13**, 254.

After reviewing past and current opinions on the subject and briefly referring to a few of the recent investigations relating to the inheritance of insanities, the writer concludes that feeble-mindedness is generally of the inherited type and that the inherita[illegible] usually recessive. Most often a single recessive gene appears to be a[illegible]ived; but, as with other abnormalities, occasionally the inheritance is of a different type. It may involve, according to the evidence in various pedigrees and investigations, (1) a recessive autosomal and a recessive sex-linked gene (Sjögren's pedigree); (2) multiple recessive successive genes; (3) a dominant gene. The insanities appear to show a dominant inheritance much more frequently than feeble-mindedness does. In dementia præcox, where the evidence seems clearest, the results are less extensive and satisfactory than for feeble-mindedness. But again in some pedigrees it is probably dominant; two genes may be involved in some, one of which may be dominant and the other recessive. In manic-depressive insanity a dominant inheritance is indicated, at least in some pedigrees. There is no reason for doubting that all forms of mental defect and aberration will be found to follow definite Mendelian rules when more extensive pedigrees have been collected and analysed. While the environment and other elements of the inheritance no doubt play a part in modifying their expression, there is no reason to believe that these induced modifications are so great that the laws of inheritance for each type of mental defect cannot ultimately be made out.

C. S. R.

[180] **Psychic impotence.**—Ben Karpman. *Psychoanalytic Review*, 1933, **20**, 274.

Barring a certain number of cases with a physical basis, the larger number of cases are psychical in origin, and the condition should be regarded as a hysterical conversion reaction. Impotence is defined here in a functional sense as any disturbance that follows interruption or interference with any part of the sexual act. It may therefore relate to the disturbances of libido, erection, pleasure during the act, ejaculation, orgasm, and the after-effects. The functional nature of impotence becomes evident when it is observed that the condition may be present with one partner and not with another. There may be, too, different degrees and variations in potency with the same partner on different occasions. The nature of the love-object will vary the potency and an antagonizing attitude and an antipathic environment may soon bring about a disturbance in this sphere. Much of the reaction is entirely unconscious. Contrary to the prevailing notion that sexual excesses lead to impotence, while abstinence is conducive to health, the reverse opinion

is here given. They may both, however, be expressions of impotence. Fear of the consequences of masturbation—impotence, insanity, crippled progeny—is a frequent barrier for full expression of potency. Such fear is unfounded. Incestuous attachment and homosexuality are prolific sources of the disorder. Ejaculatio præcox is usually conditioned on some strong emotion such as fear and anxiety of an unconscious nature. Disturbances in orgasm may be manifested by reduction in intensity, or even its absence, lack of pleasure, presence of disgust, fatigue, and even marked pain that may radiate into regions where its ætiology will not be suspected. In the majority of cases, rational psychotherapy offers a reasonable approach and many cases can be cured or improved within a short time.

C. S. R.

[181] **A study of recidivists and first offenders of average and defective intelligence.**—MYRA E. SHIMBERG and JUDITH ISRAELITE. *Amer. Jour. Orthopsychiat.*, 1933, **3**, 175.

A FOLLOW-UP study was made of 50 intellectually average and 50 defective recidivists, and of 26 average and 26 defective first offenders. Sixty-six per cent. of the intellectually average recidivists and 70 per cent. of the defective recidivists were failures as regards further acts of delinquency. Thirty-five per cent. of the intellectually average first offenders and 39 per cent. of the defective first offenders were also considered a failure. The writers conclude that there is practically no difference in adjustment between those of average and defective intelligence either as recidivists or first offenders. The prognosis for first offenders, however, seems to be twice as favourable as that for recidivists. They also subscribe to the conclusion that the early belief that feeblemindedness was in general and by itself a very important cause of crime is not definitely substantiated.

C. S. R.

[182] **The relation of menstruation to personality disorders.**—EDWARD B. ALLEN and GEORGE W. HENRY. *Amer. Jour. Psychiat.*, 1933, **13**, 239.

IN this study of 100 cases it was found that the menstrual reactions are somewhat characteristic of the different types of personality disorders. With hypomanics the flow is regular, but tends to be more profuse and of longer duration than usual; otherwise in the manic group, with one exception, no interruption in the menstrual periods was noted. In the depressive phase the patient may continue to menstruate regularly provided she is only mildly depressed. As the reaction grows more intense there may be at first an

increase in the flow and duration and subsequently it is late in appearance, becomes scant and of short duration. With profound depression amenorhœa is the rule.

Among the subacute and chronic schizophrenes there was a continuation of menstruation, but frequent irregularities in the amount and duration of the flow. With the acute cases irregularities were specially marked with occasional amenorrhœa. Menstrual disorders were more often associated with the catatonic forms and least often with the simplex type.

In the psychoneurotic patients amenorrhœa and menstrual irregularities were fairly common. There were indications that amenorrhœa was most directly associated with painful and depressive emotions.

In the miscellaneous group with various diagnoses, including psychopathic personality, paranoiacs, toxic psychoses, alcoholism and drug addicts, more than a third presented irregularities which could be associated in some cases with marked schizophrenic tendencies. As a rule minor emotional changes could be observed for a considerable period before menstrual functions appeared to be affected. The return of menstruation seemed to be one of the indications that a readjustment was taking place.

In nearly 50 per cent. of the schizophrenic cases the gynæcologist found underdevelopment of the sexual organs or of the secondary sex characteristics. On the other hand, the sexual development of the manic-depressives with a few exceptions appeared to be normal. The endocrinological and psychological aspects of menstruation are briefly reviewed and illustrative cases are reported.

C. S. R.

[183] **On pathological lying.**—D. Wiersma. *Character and Personality*, 1933, **2**, 48.

There may be found a gradual transition from normal lying to the well-developed syndrome of pseudologia phantastica. Whereas the normal liar always acts upon a definite motive—the obtaining of personal profit being often his purpose—in pathological lying the delight taken in the producing of fictive tales in itself is enough to arouse it. The more importance this delight has in the genesis of the story-telling, the more distinctly pathological the case is ; but only when a defective distinction between fiction and reality can be demonstrated have we the right to speak of pseudologia phantastica. Though untruthfulness and the nervous temperament often go hand in hand, we must not ascribe the source of pseudologia solely to this. In pseudologic patients can be traced extreme vanity and a marked egocentric attitude of mind. There is a partial infantilism of character.

C. S. R.

[184] **Juvenile types of general paralysis.**—R. M. STEWART. *Jour. of Ment. Sci.*, 1933, **79**, 602.

ABOUT 1 per cent. of all cases of congenital syphilis develop paralysis, the sexes suffering equally. The age at onset is usually between the tenth and sixteenth years, and the duration longer than in the adult type. Stigmata of congenital syphilis may be entirely absent. A general arrest of bodily development or infantilism is common. The neurological signs differ from those of the adult type in the greater frequency of focal signs, complete immobility of the pupils, and the presence of optic atrophy in conjunction with other signs of tabes. Two types of the disease may be recognized: in one the patient has been defective from birth; in the other the symptoms appear in a child of normal mental development. Expansive and paranoid delusions are uncommon, the usual clinical picture being one of simple progressive dementia. The laboratory findings resemble closely those seen in the adult type. A spirochætosis in the cerebrospinal fluid of the juvenile paretic has been recorded. Prominent among the pathological changes in the nervous system are smallness of the cerebral hemispheres, status spongiosus of the cerebral cortex, atrophy of the cerebellar folia, and binucleated Purkinje cells. Treatment by induced malaria and arsenical preparations is seldom effective.

C. S. R.

[185] **Cholesterol: its relation to mental disorder.**—A. GLEN DUNCAN. *Jour. of Ment. Sci.*, 1933, **79**, 626.

THE author's investigations lead him to conclude that (1) blood cholesterol is lowered in certain general conditions affecting the organism as a whole; (*a*) heightened emotion and quickened psychomotor activity; (*b*) fever; (*c*) hyperthyroidism. A factor common to all these is increased metabolic rate. (2) Blood cholesterol is raised in (*a*) apathy and diminished psychomotor activity; (*b*) subthyroidism. In each of these conditions there is diminished metabolic rate. (3) The properties of cholesterol are such that when the cholesterol in blood and tissues is lowered, increase of metabolic rate is to be expected; when increased, diminished metabolism should result. The analysis of actively growing tissue supports this view. (4) Therapeutically induced diminution of blood cholesterol acts on the nervous system, in some cases at least as a stimulant, causing increased mental activity. (5) Therapeutic raising of the blood cholesterol acts on the nervous system, in some cases as a sedative causing diminished mental activity. Cholesterol is one of the controlling factors of cellular metabolism, and neurones are susceptible to quantitative changes of this substance in their environment.

C. S. R.

[186] **A research on the direct observation of the capillaries in psychiatry** (La ricerca capillaroscopica in psichiatria).—A. MARI. *Riv. di pat. nerv. e ment.*, 1932, **42**, 588.

AFTER reviewing the literature the author describes his own observations in 490 cases of mental disease. He concludes that capillaroscopy is of limited value since he finds abnormalities only in the severe forms of amentia, endocrine anomalies and epilepsy, and all these may readily be observed to show abnormalities of development in other respects.

R. G. G.

PROGNOSIS AND TREATMENT

[187] **Spirochætal findings in the brains of paretics treated with malaria.**—NICHOLAS KOPELOFF and NATHAN BLACKMAN. *Amer. Jour. Psychiat.*, 1933, **13**, 21.

SPIROCHÆTES were found by the Jahnel method in the brains of six out of 39 malaria-treated general paralytics. Among the six positive findings degenerate forms of spirochætes were encountered in more instances than normal forms. Three of these patients came to autopsy shortly after malarial inoculation (within six weeks). Spirochætes were found by the Jahnel and Dieterle methods in eight out of ten general paralytics not treated with malaria and none was found in a normal brain. The validity of the technical procedure being established, it may be stated that within the limitations of the material at hand it appears that (*a*) artificially induced malaria is likely to destroy spirochætes in the brains of general paralytics; or (*b*) that failing to destroy the spirochætes completely, this form of therapy alters the morphology of the spirochæte to such a degree as to render it degenerate in appearance.

C. S. R.

[188] **Somnifaine narcosis.**—ROLF STRÖM-ÖLSEN. *Jour. of Ment. Sci.*, 1933, **79**, 638.

TOXIC risk has hitherto made somnifaine treatment an imperfect form of therapy in mental disorder. The mortality in a large series of cases collected by Müller was 5 per cent. The fact, however, that by prolonged narcosis psychotic symptoms can be allayed not only temporarily, but often permanently, is in itself such an advance that it warranted a search for modified methods which would diminish or abolish toxicity. Here a modification of technique has been described by which it is possible to eliminate dangerous toxic manifestations of somnifaine narcosis.

Toxic symptoms which frequently arise are fully discussed and their

significance pointed out. Of these the most important are ketosis, with diminished glucose tolerance, fall of blood-pressure, tachycardia, circulatory collapse, oliguria, albuminuria, disturbances of coordination, including difficulty in swallowing, pyrexia and skin eruptions of an erythematous type. Other changes to be noted are leucocytosis, fall in blood calcium and slight rise in the blood urea.

A disturbance of the carbohydrate metabolism of vital organs occurs during prolonged somnifaine narcosis—a fact not previously recorded. Clinical evidence of this is seen in the frequent development of ketonuria which has been regarded as a contraindication for further treatment. Experiments by others have shown that narcotics inhibit the oxidation by brain, liver and heart of substances such as glucose and lactic acid, which are important in carbohydrate metabolism, the inhibitions being much greater with the brain than with other organs. Ketonuria is the outward manifestation of this disturbance, but it is claimed that other symptoms, such as circulatory collapse, can be ascribed to the same fundamental cause. The knowledge that carbohydrate metabolism was affected by narcotics led to the adoption of the administration of insulin and glucose during narcosis with a view to eliminating dangerous symptoms. Of 46 treatments on 40 patients, 14 were carried out without this additional measure, and narcosis had to be prematurely stopped in five, four others being continued with smaller doses. In 15 treatments, insulin and glucose were given on the appearance of ketonuria and major toxic symptoms, with the result that these manifestations disappeared within 48 hours. In 17 instances where insulin and glucose were administered from the very beginning no ketosis or other serious complications arose. These results demonstrate that the narcotic treatment has been rendered perfectly safe by means of the modification described.

Myocardial degeneration, arteriosclerosis, marasmus and respiratory disorders constitute the chief physical contraindications for somnifaine treatment.

C. S. R.

[189] **Some experiments in reinforcing mental analysis in cases of psychosis.**—Clifford Allen. *Brit. Jour. Med. Psychol.*, 1933, **13**, 151.

A method is described whereby the discoveries of mental analysis can be reinforced with definite results (i.e. the production of dreams and variation of symptoms). This can be used as a method of research or as a therapy. To use it to increase the repressions on a major conflict or complex is dangerous, and should not be done indiscriminately. To reinforce an identification directly is dangerous also. Four illustrative cases are given, and it is held that the results obtained in this limited number appear to bear out Freud's asser-

tion as to the homosexual element in paranoid diseases, but do not confirm his mechanisms.

In the opinion of the writer, it is certain that if the psychotic patient is to influence himself some kind of autosuggestion is essential. Autosuggestion has, however, been tried unsuccessfully in the past and abandoned as useless. The experiments related here seem to show that the cause of the failure is that the suggestions have been wrongly applied and have been directed to increase the repressions on some powerful complex. It is indicated how they can be applied to influence complexes with safety and some certainty.

C. S. R.

Reviews and Notices of Books

Intracranial Tumours. By Harvey Cushing, Professor of Surgery, Harvard Medical School. With 111 illustrations. London: Baillière, Tindall and Cox. 1933. Pp. 154. Price 22s. 6d.

In this slender volume Professor Harvey Cushing puts the crown on his labours of years in a subject with which his name will always be associated, that of intracranial tumours. The book is based on his report given to the International Neurological Congress at Berne in 1931, and contains the results, in summarized form, of his study of 2,000 verified cases. No such vast experience has ever fallen to the lot of previous toilers in the same field of activity. The book may be said to present the cream of the subject, for it provides a series of tabular statements bearing on each of the principal varieties of cerebral tumour, as classified by himself, ranging from glioma (and its variants) to a miscellaneous group, there being 11 in all. The value of the work as a personal record is greatly enhanced by the tables of operative statistics—mortality percentages year by year and in gross, percentages for separate tumour groups, and so on; standards of computation are assessed and the factors governing mortality-rates examined. As a single illustration, in 1922–23, 94 patients were submitted to operation, with a case-mortality of 23·4 and an operative mortality of 16·9 per cent.; in 1930–31 the number operated on was almost double (170), but the case-mortality fell to 8·8 and the operative mortality to 6·8 per cent. These figures constitute a truly impressive record of improvement and success.

Those who are old enough to recall the times of the pioneers in cerebral surgery will be the first to appreciate at its full worth the magnificent advances made by Cushing; his work really marks an epoch in the history of that subject, and to follow its evolution during the last three decades has been a fascination and a stimulus. If he so chooses, he may well rest on his laurels, for at home and abroad his attainment is acknowledged by all.

Les Tumeurs de la Loge Cérébelleuse. By Th. de Martel and J. Guillaume. Paris: Gustave Doin et Cie. 1934. Pp. 454. Price 90 fr.

The output of special monographs dealing with one or other division of the vast subject of intracranial tumours shows no sign of diminishing. This latest contribution deals with the symptomatology, diagnosis, and treatment of growths in the posterior fossa and is based on a study of 139 examples. The following classification is adopted: tumours of the median line, cerebellar hemispheres, pontocerebellar angle, and pedunculobulbopontine axis; and

cystic arachnoiditis, respectively. Each of these subdivisions is discussed at length from a clinico-anatomical standpoint. More than half of the book is concerned with the surgical technique adopted and gives a précis of some 56 selected cases. From a neurological point of view the descriptions are adequate. Probably the chief appeal of the work is to the neurosurgeon, since procedures elaborated by M. de Martel are described at length, and minutely illustrated. It would have been interesting to find a record of the results obtained by operation, but none is supplied.

Benign Tumours in the Third Ventricle of the Brain. By Walter E. Dandy, M.D., Adjunct Professor of Surgery, The Johns Hopkins University. With 120 illustrations. London: Baillière, Tindall and Cox. 1934. Pp. 171. Price 22s. 6d.

ALL neurologists will be interested in Professor Dandy's study of colloid cysts of the third ventricle, a rare and somewhat peculiar condition that has engaged attention whenever it has appeared. Certain other benign tumours of the same region are also included in this study, which covers signs and symptoms, diagnosis, pathology and treatment in a comprehensive way. The monograph is handsomely illustrated, there being almost as many pictures as pages. Thanks mainly to ventriculography, of which the author has been the prime exponent, to whom all credit is due, the recognition of these tumours has become less elusive, and this prerequisite to surgical attack has had the consequence of facilitating the latter. In Professor Dandy's hands remarkable curative results have been secured, for which he deserves sincere congratulations. In all, some five cases of the first group and 16 of the second are described in the fullest detail; their various signs and symptoms are compared and contrasted, and tabular statements make the information easily accessible. Neurological signs cannot be depended on for diagnosis, the most usual yet non-specific being loss of pupillary reflexes; accompanying cerebellar signs are 'falsely localizing.' Diagnosis was effected by ventriculography in all of the five colloid cysts and in 12 of the 16 others; neurological examination sufficed in one alone of the whole collection.

Chirurgische Neuropathologie. Bd. I.: Die peripherischen Nerven; Bd. II.: Das Rückenmark. By Professor Dr. L. Puusepp, Tartú. With many illustrations. Tartú: J. G. Krüger. 1932 and 1933. Pp. 662 and 679. Price 35s.

PROFESSOR PUUSEPP'S work in neurosurgery and neuropathology is well known beyond the confines of his native Esthonia, and in these two compact volumes he has managed to compress a great amount of learning in regard to both peripheral nerves and spinal cord from the standpoint of present-day neurological surgery. They are excellently illustrated and richly annotated

with references. The first volume begins with a general description of individual nerves, cranial and peripheral, and of the surgical approach to each; the general symptomatology and diagnosis of peripheral nerve lesions are then examined; and the rest of the study is devoted to the symptoms and treatment of (1) traumatic injuries, (2) tumours, and (3) neuralgic states, of the same structures. The second volume follows more or less similar lines, a great deal of attention being given to contemporary technique in spinal cord diagnosis. There is much in this second volume of orthopædic interest, e.g. treatment of affections of the spinal column, as well as a detailed account of that of congenital anomalies of cord and spine. The discussions in respect of spinal tumours are full of interest and value, based as they are on personal experience. Both volumes comprise a series of statistical tables which will prove useful to the specialist, and are completed by a generous index.

The merits of the work consist chiefly in the fact that the writer is neither a surgeon turned neurologist nor a neurologist who fancies his skill as a surgeon, but one who belongs to a select coterie, trained in both schools and conversant with both aspects of his subject. This contribution to surgical neuropathology is sure of wide appreciation.

The Surgery of the Sympathetic Nervous System. By George E. Gask, F.R.C.S., Professor of Surgery, University of London; and J. Paterson Ross, F.R.C.S., Reader in Surgery, University of London. With 13 plates and 30 other illustrations. London: Baillière, Tindall and Cox. 1934. Pp. 163. Price 16s.

THE authors of this book have derived their material and experiences from work in the surgical unit at St. Bartholomew's Hospital, and have compiled a useful and interesting record, not too elaborate on the one hand or superficial on the other; and while anatomical and physiological data are properly introduced they are not made too complex for ready understanding. A few simple diagrams illustrate this part of the book. Its main divisions refer to sympathectomy for (1) disorders of the circulation, (2) disorders of visceral motor mechanisms, and (3) pain. In all of these the still experimental character of much of the treatment is duly emphasized, the importance of selecting suitable cases stressed, and the caution with which results should be interpreted underlined. But though judgment is thus at times suspended in a scientific spirit, the whole record is one of an encouraging kind, despite failures—not overlooked, but analysed. Very wisely attention is directed to the fact that 'surgical experiments' may take months or possibly years to yield their results, and that immediate issues may not represent the true sequel to sympathectomy. Among the conditions here discussed from this particular surgical standpoint may be enumerated Raynaud's disease, thromboangeitis obliterans, senile and diabetic gangrene, chronic ulcers, leprosy, megacolon, hydro-ureter and other urinary affections, dysmenorrhœa, angina pectoris, and causalgia.

Pink Disease. By Ch. Rocaz, Honorary Physician, Children's Hospital, Bordeaux. Translated by Ian Jeffreys Wood, M.D. With 20 illustrations. London: Martin Hopkinson. 1933. Pp. 153. Price 7s. 6d.

THE monograph on pink disease (or infantile acrodynia, as it is sometimes misleadingly termed) written by Dr. Rocaz gives the reader an up-to-date presentment of knowledge in regard to a curious affection whose precise nature is still indeterminate. Its history, symptomatology, clinical types, and pathology are described at some length and in a satisfactory manner; but when we turn to the question of ætiology and pathogenesis we realize how inconclusive are the opinions of the day. The author advances evidence which in his view suggests that the disease is an 'inflammation' of the whole nervous system, a 'neuraxitis,' closely allied to epidemic encephalitis. The pathological findings, however, are too variable and inconstant for this view to be substantiated; secondary infections are not uncommon, but whether any are primary is much more debatable. No pathogenic theory hitherto offered suffices to cover the whole range of disorders of mental, nervous, alimentary, cutaneous, and glandular nature; Dr. Rocaz, we think, undervalues the significance of the fact that disturbance in the vegetative nervous and reticuloendothelial systems is frequently observed.

The translation reads well; the book has good illustrations and a useful list of references.

Les Arachnoïdites Spinales Adhésives. By Dr. Dem. Paulian, Bucarest, and Dr. D. Turnesco, Paris. Paris: Masson et Cie. 1933. Pp. 96. Price 24 fr.

A SATISFACTORY little monograph on spinal arachnoiditis has been written by Drs. Paulian and Turnesco, in which its features are succinctly described, diagnosis and treatment being examined in some detail. The condition is defined as a combined radiculospinal syndrome, of variable evolution, often long drawn out or lingering, which shows evidence of being based on inflammatory reactions in the leptomeninges, with partial or complete block of the subarachnoid system. Among the causes a place is assigned to trauma, fevers and exanthems, gonococcal or meningococcal infection, tubercle (not, however, syphilis) and Kümmel's disease. The X-ray findings after lipiodol are well illustrated. Operation is recommended; of 100 cases a cure followed in 40, considerable relief in 20, and slight in 10.

Sémiologie du Sommeil. By Dr. Auguste Tournay, Paris. Paris: Gustave Doin et Cie. 1934. Pp. 136. Price 30 fr.

DR. TOURNAY has written a thoughtful essay on the phenomena of natural sleep as observed in infant and adult, and has endeavoured to explain them by reference to current views on the physiology of sleep. Three principal

modes of interpretation are scrutinized: (1) the rôle of inhibition; (2) the part played by a regulating mechanism; (3) the action of the vegetative nervous system. Of these the two first concern physiological levels, the third a physiological system. The author makes a synthetic contribution to the subject, based on these differing views, which is worthy of note. His essay is a model of logical thinking and lucid writing, replete with personal observations, and throws light on a subject of perennial interest.

Psychoanalysis and Medicine. A Study of the Wish to Fall Ill. By Karin Stephen, M.A., M.R.C.S., L.R.C.P. Cambridge: University Press. 1933. Pp. 238. Price 8s. 6d.

THIS book supplies a definite need. It is a plain and straightforward exposition of the psychoanalytic theory written principally for medical students but in non-technical language, so that even those who know nothing of medicine may be interested.

Dr. Stephen has divided her material into eight chapters, each with a summary. The first six give a brief outline of how emotional growth may become arrested as a result of the entanglement of infantile sexuality with the non-sexual instincts of fear and hostility, which have been aroused by disappointment and frustration in early life. The technique of free association is explained—that technique devised by Freud for getting into touch with the repressed part of the self in order that the affects that have caused the illness may become ventilated and dispersed. In these chapters the common criticisms of psychoanalysis are dealt with in a helpful manner. Psychosexual development is traced through its several phases and the factors that may constitute towards fixation at each level discussed.

The most important chapters in the book are possibly those which deal with anxiety and guilt and with the mechanisms adapted by normal individuals—by sublimation—and by those abnormal—by symptom formation—to defend themselves against these disturbing influences. Characteristics of the primitive unconscious are pointed out: (1) Its complete disregard for consistency, (2) its belief in magical thinking and wishing, (3) its confusion of fantasy with fact and of thinking with doing. The author touches upon the vexed question of the transference by means of which psychoanalytic therapy progresses. Almost the last paragraph in the book shows the necessity there is for the analyst himself to have been psychoanalysed before dealing with others by the same method. Dr. Stephen's book does not suffer, as do so many works dealing with this subject, from being too highly theoretical or too clinical. The outstanding advantage is that she has contrived to combine the theory—clearly and simply described—with well-chosen clinical material.

A. C. W.

A Handbook of Psychiatry. By John H. Ewen, M.R.C.P. (Ed.), D.P.M., Assistant Medical Officer, Surrey County Hospital, Netherne. London: Baillière, Tindall and Cox. 1933. Pp. 267. Price 12s. 6d.

THIS useful volume presents in the form of a synopsis the principles of modern psychiatry and epitomises the more recent work on the subject. We agree that owing to lack of knowledge regarding ætiology and pathology in many forms of mental disturbance no really satisfactory classification of mental diseases has yet been attained. The main classification adopted is that of the Clinical Psychiatry Sub-Committee of the Royal Medico-Psychological Association, 1932. Other classifications are also discussed briefly, including the ætiological (Clouston and Tuke), the physiological (Wernicke), the psychological (Pritchard, Helmroth and Ziehen), and the symptomatological; also that of the Board of Control, 1932. The chapter on ætiology is well arranged and includes summaries of the views of Freud, Jung, Kempf and Adler. As regards heredity, no neurologist will agree with the statement that statistics indicate that epilepsy shows an inherited taint in 80 per cent. of cases and that transmission is frequently direct. It is not quite clear if this is intended only to apply to gross epilepsy with psychotic changes, for it is certainly not true of epilepsy considered as a whole. The bodily types differentiated by Kretschmer are summarised and delusions and hallucinations well analysed. Separate chapters deal with disorders of memory and insomnia, including narcolepsy. The doubtful ætiology of schizophrenia is emphasized by the fact that no less than nine theories of causation are mentioned, ranging from a habitual tendency to introspection (Jung) to focal sepsis (Cotton).

Paraphrenia is rightly accorded a separate section. The absence in many cases of a constitutional basis and of previous mental instability, as well as the prominence of the characteristic symptoms, justifies the separation of involutional melancholia from the melancholic phase of manic-depressive psychosis. It is interesting to note, in view of a recent pronouncement, that no mention of drugs of the barbituric series is made in the section dealing with 'psychoses due to drugs.'

Chronic hallucinatory psychosis, defined as a prominent hallucinosis with little disturbance of mind in other directions, is dealt with separately, although it is admitted that some difference of opinion exists as to its being a clinical entity. A valuable chapter is that dealing with 'psychosis associated with physical disorder.' It is interesting to find the definite statement, a fact of which few are aware, that in Addison's (pernicious) anæmia mental symptoms may occur before any anæmia is manifest. The estimation that mental symptoms occur in 60 to 85 per cent. of cases of cerebral tumour appears far too high and is evidently judged more from a psychiatric than a neurological standpoint. The statement that in the acute stage of encephalitis lethargica the cerebrospinal fluid, among other changes, 'rarely shows'

a positive Wassermann reaction is very misleading; otherwise, the features of this disease are well described. The alternative classifications of the psychoneuroses (Freud, Janet, etc.) are discussed and the main features of each variety epitomised.

A section is devoted to Mental Deficiency and a further one to the important subject of the legal relations of mental disorder. In the latter the McNaughton ruling of 1843 is reviewed as well as the civil responsibility of the insane. A useful paragraph is that on the examination of patients accused of a crime.

This book can be strongly recommended as a handy work of reference, and to those who wish to become familiar with modern views and nomenclature in psychiatry but who cannot spare the time to read the larger textbooks. Also, it will be valuable especially for revision purposes to those reading for a Diploma in Psychological Medicine and similar examinations.

Personality, Many in One. By James Winfred Bridges. Boston: Stratford Co. 1933. Price $2.

THE author has taken a ramble in the fields of psychology, and *Personality, Many in One*, describes the book exactly.

In his travels he has wandered from simple facts of academic psychology as far as the neuroses, Jung's types and psychoses. He bases his work on the assumption that the primitive character lacks organization and consequent direction, and lays stress on the importance of synthesis, and organization of the various mental components. Interference and integration and undirected organization give birth in his opinion to the embryo of psychopathology. As far as we can make out, the author disagrees with the concept of the unconscious according to Freudian interpretation, and this disagreement is probably responsible for the absence of reference to Freud. He lays emphasis on the fact that there is no gap between the normal and abnormal, but that they grade into each other by imperceptible degrees. He also points out that mental ability is not determined by quantity of cells and tissue, but rather by neural plasticity and ability of building up a complex organisation. He believes that the idea of character and successful synthesis of personality is the persistence of motivation, which is the outcome of organisation, a product of training and experience. He refers to the work of Berman and MacAuliffe on the importance of the endocrine factor in the successful integration of personality, and mentions the work of Robert Sandek on the importance of graphology in diagnosis of character. As the author truly states that any book is good to a reader if he can abstract something useful from it, this book should prove of value to the student of psychology. There is a useful index, and a copious bibliography.

The Treatment of Functional Nerve Cases by the Method of Neuro-Induction. By Leonard Inkster, M.A., M.R.C.S. London: H. K. Lewis and Co. 1933. Pp. 73. Price 3s. 6d.

NEURO-INDUCTION is here presented to the general practitioner, who may require to treat 'functional' cases as a method 'asserted to be both powerful and almost invariably pleasant to the patient.'

The technique is simple: total relaxation of mind and body is secured; and this is accompanied by a 'hyperconsciousness of the mentality.' The operator by suggestion and explanation then introduces 'philosophic considérations' calculated to induce ease in thinking. Ease in thinking is right thinking, we are told, and is 'logically bound to be so because our whole analysis of what is right thought is based on discovering what thoughts make men's minds easy.' The logic or value of this method is not immediately obvious. It bears a striking resemblance to hypnotic treatment, although Dr. Inkster distinguishes the two. He adds, however, that 'we need not bother very much about whether neuro-induction might or might not possibly be called a sort of hypnotism.'

E. A. B.

Forty Years of Psychiatry. By William A. White, M.D., Sc.D. New York and Washington: Nervous and Mental Disease Publishing Company. 1933. Pp. 154. Price $3.

DR. W. A. WHITE is a distinguished American psychiatrist, many of whose writings are well known to physicians in this country. In the present volume he has aimed at giving a picture of the progress and advances in psychological medicine in America from the beginning of the present century. The result is a stimulating book which should be read by every worker in this field.

Within the compass of 154 pages the author draws clear-cut pictures of all the principal advances; thus, the transition from the humanitarian to the scientific period, the modern mental hospital, the advent and growth of psychoanalysis, the increasing acceptance of the psychological factor in general medicine, mental hygiene and child guidance clinics are but a few of the developments described. Dr. White is peculiarly well fitted to write such a book since he himself has played a conspicuous part in many of the advances. With Dr. Jelliffe he launched *The Psychoanalytic Review* in 1913 when the number of adherents to that movement were few. Any development which promised a better understanding of the nature of mental illness or mental health has always had the advantage of the author's powerful advocacy. In regard to the future he expresses himself as confident that psychological medicine will continue to present in its results some important and significant additions to scientific knowledge.

J. F.

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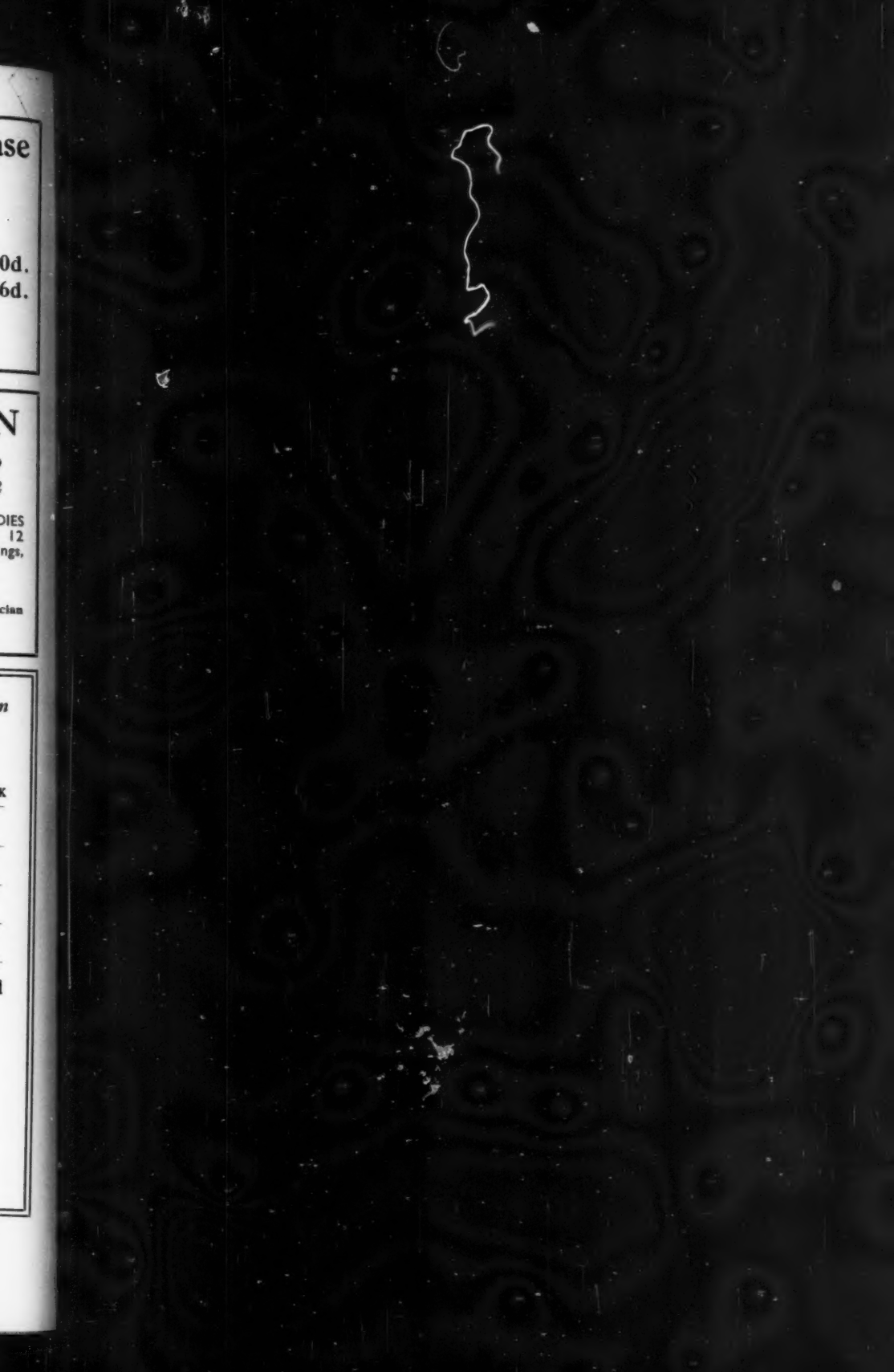

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